

AMERICAN JOURNAL OF PSYCHIATRY

HALLUCINATIONS; THEIR NATURE AND SIGNIFICANCE.*

By C. MACFIE CAMPBELL, BOSTON.

Among the various symptoms with which the psychiatrist has to deal hallucinations play a prominent rôle. They form a dramatic element in many a clinical picture; they may dominate the behavior of the patient; they are of considerable diagnostic and prognostic significance. In the usual case record one finds a reference to the presence or absence of hallucinations; "hallucinations of sight," "hallucinations of hearing," may be summarily entered on the record as if these terms were as definite in their connotation as fever or leucocytosis. Theories as to the origin of hallucinations are sometimes propounded without a thorough review of the actual phenomena which are covered by the general term. As a matter of fact, when one proceeds from the word "hallucinations" to the actual clinical material one finds a much more varied picture than is suggested by this one word. In some cases the patient behaves in exactly the same way as he would under the influence of an ordinary sense perception in response to an external stimulus. He reaches out to grasp something which the physician cannot see; he points with staring eyes at imaginary animals; he flees in horror to the police station to get protection from threatening voices which terrify him; he shouts out abuse in answer to the abuse which he hears, plugs his nose with cotton wool to keep out disturbing smells, spits out the distasteful material which he feels in his mouth. As far as the behavior of these patients is concerned, it would seem to be the same as that determined by an external stimulus.

* Read at the eighty-fifth annual meeting of The American Psychiatric Association, Atlanta, Ga., May 14, 15, 16, 17, 1929

In other cases the behavior of the patient does not suggest the assumed presence of an actual external stimulus. The patient tells of hearing voices but neither answers the voices nor seems to be especially affected by them; he does not turn his head nor specify the exact quarter from which the voices come. The patient claims to have seen certain objects but finds difficulty in giving specific details as to form, color, distance, and his external behavior may have given no hint of the special experience. When questioned about the latter the patient may be conscious that the experience, which he has formulated in terms of "hearing" or "seeing," did not have the same quality in relation to reality as is usually connoted by these terms.

Not only may the experience lack the reality value of the normal sensory experience, but when accurately studied it may prove to be something difficult to formulate in the terms of normal experience. The patient, when asked about the voices heard, may indicate that they are not voices heard in the usual way, they are not heard through the ear but they are heard in the head. They come in a special way. The differentiation between voice and thought may not be very clear and in the formulation of the experience the patient may oscillate between having heard a voice and having had a thought. In some cases the term "voice" is clung to by the patient but it has a peculiar setting; thus the patient may hear the voice coming from the heart or the abdomen.

A pedlar, 45 years of age, complained of a small being of female sex in his abdomen: "Its voice is high pitched. . . . I can hear its voice. . . . It can talk just as plain, ask questions just as smart . . . for two years it has done nothing but nag and nag. . . . It scolds me if I try to drink more than one cup of coffee."

The experience as formulated by the patient may be so phantastic that the physician can see no analogy between it and the structure of his own experience.

It is beyond the scope of the present communication to make a systematic survey of the whole field of hallucinatory experiences; the aim is rather to emphasize the diversity of the phenomena and to illustrate some of the processes involved.

In classifying hallucinations in general, one may consider their personal value or significance. Some hallucinations are like casual

sights and sounds which have no special relationship to personal interests, emotions or experiences; they appear in the patient's field of experience in a quite irrelevant manner, and do not seem to have borrowed their structure or quality from special preoccupations of the individual, either overt or repressed. This is frequently the character of hallucinations which owe their origin to impersonal factors, organic or toxic.

A man of 28 complained that for two months he had been seeing objects in front of him such as a cow, a cat, a dog; the objects appeared to be at a distance of eight or nine feet and about five feet from the ground; "they look real like a picture but I always think, well! you can't have a cat or dog in the air." The neurological status gave no unequivocal evidence of brain tumor or other form of organic brain disease, but the patient had been subject to headache, sleeplessness, restlessness, dizziness, vomiting.

In other cases the nature of the hallucinations has obviously been determined in part by the special domestic or occupational interests of the patient, as in the familiar occupational delirium, or by individual traits of the patient, such as his special sensory endowment. Thus Schumann the composer during his serious psychosis would hear whole orchestral pieces; in earlier attacks every noise he heard was converted into musical sounds.

In an important group of cases the hallucinations are not extraneous or irrelevant sensory elements in the periphery of the personality and its experience, but are closely related to intimate matters of great emotional value, such as thwarted ambitions, personal attachments, religious aspirations, sexual conflicts, painful memories.

One may discuss in a little more detail the two main groups referred to, the group of the more peripheral and impersonal hallucinations, and that of the more intimate and personal hallucinations.

A. Hallucinations of simple and impersonal type may be caused by acute or chronic intoxication with a variety of drugs. The subjective sensory experiences due to the administration of "mescal button" or Peyote (*Anhalonium Lewinii*) have been studied in some detail by a variety of authors. Weir Mitchell¹ in 1896 published his personal experience with the drug, and recently extensive monographs have been devoted to the subject by Beringer and by Roubier; a brief and interesting review of the topic has been

written by Kluever.³ Beringer⁴ describes in considerable detail the reaction of various experimental subjects to the drug; there was considerable variation from subject to subject; the subjective sensory experiences seemed to have little relation to the special interests of the individual; they were recognized as subjective although very vivid; only in exceptional cases did the subject take them for objective reality. Various senses were involved; one subject smelled vague perfume, in others the taste was modified; noises were modified so that an auto sounded like an orchestra; scratching, snapping, scrunching noises were heard; one subject on striking a single note heard a choir of female voices. As to the visual experiences observed, peculiar colors lay on objects, stereoscopic changes were noticed, objects appeared in movement, there was spontaneous elaboration of scenes of perceptual vividness. Synæsthesiæ were frequent; thus, the stroke of the clock elicited a purple color, the noise of winding up the clock caused a feeling as if the arm were put in screws. The drug caused the special elaboration of actual sense stimuli, but also the elaboration of sensations produced centrally by the drug. In the acute hallucinatory psychoses of the chronic alcoholic one meets many of the symptoms referred to above; the appreciation of the subjective character of the sensory experiences is, however, soon lost and the hallucinations are more apt to be colored by personal elements, sometimes of less intimate nature such as occupational interests, sometimes more intimate and frequently in relation to the sex life of the individual.

As the hallucinations produced by the introduction of a drug into the system may betray little of the personal element, so it is with the hallucinations due to the coarse interference of a neoplasm. Horrax⁴ in 1923 reported on visual hallucinations associated with organic disease of the brain; these hallucinations varied from elementary light sensations such as a scintillating scotoma through perceptions of colors, figures, or shadows to the vivid perception of definite objects. In such experiences the appreciation of their subjective nature is, as a rule, retained unless there is some general clouding of consciousness. In the discussion of this paper S. A. K. Wilson contrasted the crude hallucinations associated with injuries to the receptive areas of the occipital or temporal lobe with the more highly organized hallucinations due to damage to the association areas.

In discussing hallucinatory experiences of simple and impersonal nature mention must be made of the hallucinations occurring in cases of cardiac and pulmonary diseases to which Henry Head⁶ called attention in 1901. In patients with these forms of visceral disease Head noted the occurrence of hallucinations of vision, consisting of draped figures white, black, or gray but never colored, usually stationary, neither arms nor legs being seen. These hallucinations remind one of Weir Mitchell's mescal experiences; on awakening he would see the door open and a procession of white-robed, veiled figures enter. Head's patients were also subject to simple auditory hallucinations, with whispering, tapping, knocking, ringing; unpleasant odors were also experienced.

While hallucinations may be caused by special toxins or by gross intracranial lesions, the constitutional endowment of the individual may play an important rôle in their development. We are indebted to Galton⁷ for emphasizing the great difference that exists in the mental imagery of different individuals. To some of those questioned by him the term visual imagery had practically no meaning, while others described their visual imagery as of unusual vividness. He refers to those who can read off mentally the scores of music or manuscript, and quotes Mr. Flinters Petrie who could work out sums with the aid of an imaginary sliding rule.

Galton discusses in some detail synæsthesia, the phenomenon of the association of a quality of one sense with the stimulus of another sense; thus in some people musical notes give color experiences, taste may call up a sensation of color, *e. g.*, a sour solution may give a sensation of green. The synæsthesiae referred to above as occurring after the action of mescaline may be largely determined by the constitutional endowment of the individual subject of the experiment. Further examples of synæsthesia may be found in an article by Wells⁸ on Symbolism and Synæsthesia. Other interesting examples of subjective sensory experiences are referred to by Galton; Goethe said that "whenever he bent his head and closed his eyes and thought of a rose a sort of rosette made its appearance which would not keep its shape steady for a moment but unfolded from within, throwing out a succession of petals, mostly red but sometimes green." The frequency of hallucinations in people who otherwise are perfectly normal and efficient is emphasized by Galton; an authoress saw the principal character of one of her

novels glide through the door straight up to her; the daughter of a musician often imagined that she heard her father playing. "In short the familiar hallucinations of the insane are to be met with, far more frequently than is commonly supposed, among people moving in society and in good working health."

The recent work of the Marburg school of psychologists on visual imagery brings data from a special field, which illustrate the general topic of the individual variability of mental imagery. Some individuals can do more than merely imagine clearly a visual experience, they can revive the earlier optical impression with hallucinatory clearness. This type of mental image is called an "eidetic" image (Gr. *εἶδος τό*, that which is seen). The eidetic image is perceptual, it is "seen," it has external localization, it is more detailed than the ordinary image, it is subject to voluntary recall. "Eidetic imagery is a common possession of children; the ability usually retreats with advancing age, but vestiges often remain in later life, and well-marked cases of the *eidetische Anlage* are met with among adults" (Allport⁸). This eidetic endowment may be an important asset to the literary artist and Kroh⁹ has claimed that certain German authors can be diagnosed as "eidetics"; among French authors Balzac, if the account of Benjamin, his biographer, is well founded, would stand out as a typical example. As this endowment may play an important rôle in the creative work of the literary artist, consciously moulding his work according to artistic requirements, so, in the reactivation of childish modes of experience in the adult psychosis, it may take its place along with other childhood tendencies, cravings and formulations and in part determine the sensory vividness of the hallucinatory elements.

In the mescaline experiences the response of the eidetic is claimed to be somewhat different from that of the non-eidetic, but, on the whole, the constitutional endowment of the individual plays here a subsidiary part. In these sensory experiences, as in the visual hallucinations of alcoholic delirium and of brain tumor, physiological conditions and bio-chemical changes seem to be the main determining factors. The fact that in the alcoholic delirium the hallucinations are predominantly visual cannot be attributed to the disproportionate alcoholism of the eidetic, but depends no doubt on the special sensitiveness of the visual system to the metabolic products responsible for the delirium.

When one passes from the simple hallucinations discussed above to the hallucinatory experiences that occur in the setting of the functional psychoses and of complicated emotional conditions, one can no longer think merely in terms of toxic and structural factors and of special sensory endowment but has in addition to take into account the cultural background, the rôle of suggestion, the complexity of the emotional life, the rôle of desire and of the creative imagination. A simple illustration of the complex situation may be given in the following case, chosen almost at random:

The patient, a girl of 19, was much preoccupied with thoughts of her mother dead eight years previously, and of a sweetheart with whom, during the past year, she had been indiscreet. She had heard her mother's voice telling her to go where her sweetheart lived. The patient stated quite clearly that she had heard her mother's voice; the voice came from right beside her; "I didn't think she was there, I thought she was talking to me," but again she denied that she thought her mother was there. She admitted that a voice cannot exist by itself; her only explanation of the experience was, "I was thinking of her at the time." Such statements seem to indicate, on the one hand, a definite perceptual quality of the experience, but, on the other hand, a certain appreciation of the relationship of the experience to her own preoccupations. The patient does not make a consistent integration of different attitudes, but allows them to exist side by side; on the one hand, an animistic attitude with vivid perception or dramatization of the voice, on the other hand, the critical realization of the psychological situation at a much more rational level.

Such a condition is not fixed and static; there is an oscillation between the different levels of thought and there are intermediate stages where prelogical and phantastic elements mingle with realistic and adaptive formulations, while the perceptual quality of the experience may be hard to evaluate. The hallucinatory element in such a clinical picture is something very different from the insects which the delirious alcoholic sweeps off his coverlet, and from the brownies which the fever convalescent may languidly watch as they perform their antics on his bedstead.

In the following case one sees a somewhat similar type of experience where a dominant preoccupation leads to misinterpretation

of things seen and heard, and occasionally to an actual hallucinatory experience.

The patient, a woman of 47, had been deserted by her husband; her boy was living in another country. After her husband's disappearance she often thought that she heard his voice in various places, and occasionally she believed she saw him in the distance. In the hospital when a patient cried she said, "I thought I heard my boy crying." In this case as in so many others the experience represents the simple and direct realization of an experience, ardently and consciously desired. In a series of such cases the degree of the perceptual quality varies within wide limits, and is influenced by such factors as the original endowment with imagery, the environmental conditions, the alertness or drowsiness of the patient, the emotional tension, the greater or less involvement of the critical faculty. It is only to certain individuals and under certain conditions that there is granted the autistic gratification of the wide-spread longing

"But oh for the touch of a vanished hand
And the sound of a voice that is still!"

The preoccupations which are at the basis of hallucinations are frequently of sexual nature; in many cases they are concerned with the personal worth of the patient, and with a tendency towards compensation of religious or other type. A woman of 39, who had lived an irregular sexual life, became somewhat moody. One day she heard the voice of God say "you cannot serve two masters"; and again, after spending much time in religious observances, she heard a voice say "I come to you in a Carmelite garb." Such an experience is a familiar one in the religious field, with wide variability as to the perceptual quality and as to the acceptance of it by the personality. The "still small voice" of conscience to most of us has lost its perceptual quality, and does not address us so abruptly as the voice which said to Paul on the road to Damascus, "Saul, Saul, why persecutest thou Me?"

In the following case the perceptual quality was outstanding. The patient, a hobo of 26, presented a picture not unlike that of the previous patient. Three years before admission to the hospital he had heard a voice say "go to confession, go to confession"; more recently a voice said, "it is too late for you to repent." During an interview with the physician a voice said to him "don't talk, don't

talk, keep praying"; after a moment he said to the physician "to H—I with the voice, I will answer your questions, go ahead." From such cases where the hallucination seems to have a definite perceptual quality, if not a quality identical with that of the normal sense experience, there is an uninterrupted gradation to others where the experience seems to pass entirely at the ideational level, but where there is a definite objective reference.

In the life of Saint Teresa of Spain we read of experiences, described in the language of the senses, but in which the absence of any sensory component is emphasized and the process is stated to have occurred at the ideational level. She heard a Voice say, "I would have thy conversation not with men but with angels," and thereupon she broke off her worldly friendships. In the words of her biographer:*

These voices, she says, were not discerned by the bodily ears; yet they sounded clearer than ever was voice heard by the outward ears. Ineluctably heard they were; for the ears of the soul cannot be stopped like the ears of the body. Nor can they be supposed something imagined; for though an imagination might be mistaken for the Other Voice, yet the Other Voice when it comes is altogether different, and brings with it a majesty that is its own credential, a compelling power which leaves the soul holier and stronger.

With regard to her visions we have equally explicit statements; to quote in translation the words of Saint Teresa herself:

The day of the glorious Saint Peter, being at prayer, I saw or rather felt, for I saw nothing, whether with the eyes of the body, or yet with the eyes of the soul; but it appeared to me that Christ was by my side, and I knew it was He who used to speak with me. I was quite ignorant there could be this sort of vision and at first feared and could only weep; but when He spoke one word to reassure me, I became as I was wont quite tranquil and full of joy. It seemed to me that Jesus Christ stepped always at my side; and as it was not an Imaginary Vision I could not tell in what form.

In contrast with that "Intellectual Vision" is the Imaginary Vision described in the following passage:

Once, on Saint Paul's day, being at Mass, there was represented to me the whole of the most sacred Son of Man as He was raised from the dead, with great beauty and majesty. . . . This vision, never did I see it, nor any vision, with the eyes of my body, but only with the eyes of my soul.

One day at the Communion before she had received the Bread, she saw the Holy Dove floating over it, and heard the sound of the

hovering wings. The most famous of her visions is that one to which she owes the name Teresa of the Transverberated Heart :

Advancing towards my left side, I saw an angel in corporeal form, which was rare with me, for though often I have had perceptions of angels it has been without seeing them. He was small of stature, beautiful exceedingly, his countenance a burning flame. In his hand was a spear of gold, at its point a little flame. It seemed to me that with this he thrust through my heart, not once but many times, and it pierced to my very innermost. It left me all on fire with great love to God.

How different in their emotional intensity are such experiences from the "revelations" ascribed by tradition to Mohammed¹⁰ which were always available at a moment's notice, for authoritative answers to unexpected questions !

The hallucinatory experiences of personal nature so far discussed have been the expression of conscious preoccupations ; in other cases hallucinations are based upon trends which are more or less completely dissociated from the main personality. In the study of such hallucinations where the analysis of the content is of great interest, it is also important to pay attention to the perceptual quality and reality value of the hallucinatory factor. A more detailed review of the subject would include an analysis of the phenomena of the spiritualistic séance and of those co-conscious images to which Morton Prince¹¹ has devoted several fascinating studies. In such an analysis due attention would have to be paid to the constitutional predisposition to dissociation, the native type of sensory imagery, the capacity for imagination and dramatization, the indulgence in habitual day-dreaming, the rôle of suggestion and the individual gain from the special manifestations.

In the general category of hallucinations there may be included phenomena of less dignified nature than the hallucinations of impersonal and personal type so far illustrated. The following case is an example.

A Portuguese lad of 15, of limited intelligence (his Intelligence Quotient 65, however, was probably not representative) was brought to the hospital, as he had run away from home several times, and had stolen some money. He stated that at the age of 12 he had seen spirits walking round his bed ; they were small and appeared like skeletons. Some months before his admission to the hospital, the cow in the barn had looked at him, moved her lips and

said, "you must leave this barn and never come back here again, you will do well, you are in danger here, happiness and wealth await you elsewhere." Two weeks later the goat spoke to him and said, "never hang around this corner, it is an unsafe corner for you, you call it hell's corner." Every time he looked at the corner he would see the devil; he gave detailed descriptions of the devil. He had been ruminating over his grandmother's account of the meaning of whirlwinds, which was that evil is in the air. At times he would see angels dancing around in heaven beckoning to him.

In the hospital the suggestion that he would see the devil in a corner would bring on a dramatic reaction, and after such a scene of terror he would tell how he had seen the devil pointing to him, and saying, "you come to me." According to the patient's statements he believed that the devil actually appeared before him, and that the angels' figures were objective, while he doubted whether the farmyard animals were responsible for the words which he claimed he had heard. It is no easy matter to determine how far in such dramatizations and romances there is a perceptual element, and how far and how consistently and how consciously the individual is carried away by his imaginative creations.

Another patient, a lad of 18, had for several years been indulging in complicated sexual ruminations and activity; more recently there had been elaborate phantasies and hallucinations.

Three weeks before admission to the hospital he saw an angel. He came to the hospital because he heard voices tell him that he would go to heaven if he killed himself; he therefore walked along the railroad tracks and jumped into the harbor. In the hospital he gave many details of this experience: "I thought the Superior of St. John's Convent called over to me from the sun. She said, 'I am greater than you.' I said, 'no I am greater than you.' She said, 'if you are greater than I show yourself to the people.' I came down and showed myself to the people." He claimed that he saw angels in the hospital in the form of moving pictures, and that he saw plainly a man falling in a prone position from near the ceiling to near the ground. At one and the same time he admitted that it was only imagination and was positive that he saw the man. Here, too, as in the previous case there is much dramatization and phantastic elaboration, while the material of his ruminations and phantasies is presented under somewhat archaic forms.

In such a case it is even more difficult than in the preceding one to make any clear statement as to the perceptual quality and judgment of objectivity in his hallucinatory experiences, for at this archaic level of thought objective and subjective have not as yet attained clear differentiation and perceptual quality is a term only applicable to a less remote stage of the evolution of human thought.

SUMMARY.

In clinical psychiatry the term hallucination is used to cover a great variety of experiences.

In this paper a brief review is made of the simple and impersonal hallucinations associated with a variety of toxic and organic conditions, and of the more complex and personal hallucinations closely related to important preoccupations, which are not infrequently dissociated from the rest of the personality.

BIBLIOGRAPHY.

1. Mitchell, S. W.: Remarks on the Effects of Anhalonium Levinii (The Mescal Button). *Brit. Med. Jour.*, Vol. 2, p. 1625, 1896.
2. H. Klüver: *Mescal: The Divine Plant and Its Psychological Effects*. London, Kegan Paul, Trench, Trubner & Co., 1928.
3. Beringer, K.: *Der Meskalinrausch. Seine Geschichte und Erscheinungsweise*. Berlin, Julius Springer, 1927.
4. Horrax, G.: Visual Hallucinations as a Cerebral Localizing Phenomenon. *Arch. Neurol. & Psychiat.*, 10: 532 (Nov.), 1923.
5. Head, H.: Certain Mental Changes that Accompany Visceral Disease. *Brain*, 24: 344, 1901.
6. Galton, F.: *Inquiries into Human Faculty and Its Development*. London, J. M. Dent & Co.
7. Wells, F. L.: Symbolism and Synæsthesia. *Amer. Journ. Ins.*, 75: 481, 1918-19.
8. Allport, G. W.: Eidetic Imagery. *Brit. Jour. Psych., General Section*, 15: 99 (Oct.), 1924.
9. Colvill, H. H.: *Saint Teresa of Spain*. London, Methuen & Co.
10. Margoliouth, D. S.: *Mohammed and the Rise of Islam*. London and New York, G. P. Putnam's Sons.
11. Prince, M.: Coconscious Images. *Journ. Abn. Psych.*, 12: 289 (Dec.), 1917.

BEHAVIOR DISORDERS IN CHRONIC EPIDEMIC ENCEPHALITIS.

CLINICAL COURSE IN RELATION TO SIGNS OF PERSISTING ORGANIC
PATHOLOGY.*

By CHARLES E. GIBBS, M. D.,

*Director of Clinical Psychiatry, Kings Park State Hospital,
Kings Park, N. Y.*

This paper is based on the records of 144 patients admitted at Kings Park during the eight years from January 1, 1920, to January 1, 1929. All were admitted because of mental symptoms and were classified as psychosis with encephalitis lethargica. In 114 of these patients the onset of encephalitis had occurred before 15 years of age. Practically all of these children were admitted because of severe behavior disorder. This high proportion of children was due to special facilities having been provided for them. Soon after the appearance of epidemic encephalitis early in 1919 these difficult children became a problem to various clinics and institutions in and around New York. Many of the more severe cases were sent to the state hospitals. To give these little patients suitable care a colony was established as Kings Park in the spring of 1924. Since then this colony has operated at full capacity on a program of habit training, school work and play. It has been a distinct success, and with no elaborate outlay for personnel or equipment.

The fact that this colony of 50 children is still operating at capacity, six years after the peak of the epidemic, has drawn our attention to the potential importance of encephalitis from the standpoint of public health, mental hygiene, and hospital beds. Nearly all of these 144 patients had the onset of encephalitis before 1926, and it is striking that 84 of them still require hospital care, while only eight have been discharged recovered, and 10 have died. More than one-half of them have developed some degree of Parkinsonism, but with mental disorder persisting and keeping them in the hospital. It is evident,

* Read in Abstract at the eighty-fifth annual meeting of The American Psychiatric Association, Atlanta, Ga., May 14, 15, 16, 17, 1929.

therefore, that the surviving victims of encephalitis who develop mental disorder follow a stormy and prolonged course, the duration of which we do not yet know. It is true that the total number of these patients is small in proportion to the population of a mental hospital, but they are large in proportion to the number of persons attacked by encephalitis.

In New York State during the past eight years over 4000 cases of epidemic encephalitis were reported, and over 2000 deaths, leaving something over 2000 survivors. During the same period there were over 400 first admissions to the New York civil state hospitals with a diagnosis of psychosis with encephalitis lethargica. In other words, 10 per cent of those having the disease, and 20 per

TABLE 1.
HOSPITALIZATION OF 144 PATIENTS.*

Year	Number patients	First admissions		Readmissions	
		Continuously in hospital or on parole to April 1, 1929	Discharged, later readmitted and in hospital or on parole April 1, 1929	Number of patients	In hospital or on parole April 1, 1929
1920.....	2	1
1921.....	4	2
1922.....	5	2	2	1	..
1923.....	6	1	2
1924.....	30	11	4	2	2
1925.....	36	14	9	4	2
1926.....	28	8	4	5	3
1927.....	23	12	2	8	3
1928.....	10	10	0	13	13
Total.....	144	61	23	33	23

* Includes admissions to this and other state or recognized mental hospitals. Some had their first admission to other hospitals and were transferred or readmitted to Kings Park, and *vice versa*.

cent of those who survived, have reached state hospitals. And it seems certain that many of them will have a hospital residence of several years' duration. (See Table 1.) No other infectious disease contributes so large a proportion of its victims, or for so long a period. It is evident that in the event of a severe or widespread outbreak of encephalitis those who survive will present a serious problem to clinics and hospitals. The disease has already become a serious public health problem in England, where an exhaustive inquiry into the fate of these patients has recently been made by Parsons for the Ministry of Health.

From the clinical point of view encephalitis has been given a position of strategic importance in both neurology and psychiatry. To neurologists these patients present a wealth of pathology of

great value in working out the function of certain parts of the nervous system not yet well understood. Psychiatrists, on the other hand, find patients presenting a variety of mental and behavior reactions which are functional in type but which are evidently produced or activated by an organic brain lesion. For this reason these patients have been a source of great psychiatric interest, and there have been many suggestions that encephalitis may throw some light on the nature of psychopathic personality and the functional psychoses. Following the early suggestion of Von Economo, numerous papers have appeared on the possible relation between Parkinsonism and catatonia. That this similarity is superficial is now generally agreed by those having much to do with these patients. More striking and significant is the resemblance of the behavior disorders in children after encephalitis to the behavior disorders seen in psychopathic and so-called problem children. There is good reason to think that a better understanding of the one would lead to a better understanding of the other.

With this idea in mind the cases at Kings Park have been reviewed, but with the preliminary and primary purpose of gaining a better conception of the essential clinical picture, the underlying pathology, and the lines along which further study should be pursued. These cases seemed to be of unusual value for a study of the clinical course of the behavior disorder over a period of five to ten years from the onset of encephalitis. The observations to be presented refer to some of the clinical features of the behavior disorder, to the importance of the emotional factor, and to the clinical evidence that the behavior disorder is associated with a persisting process in the brain.

The abnormalities of behavior which these children display have been so well described and are so familiar that detailed description here is unnecessary. It seems important now, as has been indicated by Bond and Partridge, to consider whether the behavior disorder of encephalitis is characteristic and specific, and to be differentiated from that of psychopathic or problem children. While it is true that if we make a list of the individual acts of encephalitis children and compare them with the acts of psychopathic children they are similar and show the same wide variety, yet taken together they present a different picture, and the patients themselves are different. The behavior disorder of encephalitis seems to have a

fairly definite clinical course in fairly definite phases, of different duration in different patients, but apparently not determined by external influences. It is not meant to say, however, that the intensity and form of the behavior reactions may not be largely determined by external factors, or that the behavior of these children is not subject to some modification and control. Under the most favorable influences, however, the disorder seems to pursue its course to one of several ends.

As being characteristic of encephalitis, we have been impressed by the frequent history in these children of a period of extreme restless overactivity with emotional irritability and impulsiveness. This phase develops suddenly or gradually after the acute phase, and persists for months or years. These children seem to be driven by a constant nervous stimulation or irritation, are constantly annoying those about them, fighting impulsively, and running wild about the house and in the street. The similarity of this motor overactivity to manic excitement has been emphasized by Marshall. The emotional factor seems somewhat different, but this difference may be one of degree, or modified by the early age of these patients. The term "hyperkinetic" should not be used to describe this overactivity as it has already been given another meaning in encephalitis by Wimmer, referring to purely neurological motor signs, as tremors, myoclonias, choreiform and athetoid movements. As will be pointed out later, it seems doubtful whether this overactivity should be given a too strictly neurological interpretation. After a period of months or years this restless activity gradually subsides, but the emotional disorder, impulsiveness, and various other things persist.

During this second phase the behavior is more comparable to that of psychopathic children, but some fairly definite characteristics remain. Many of these children have begun to steal, lie, run away, and do most of the other things that psychopathic children do. But as compared to the psychopathic child their behavior is more simple, open, impulsive and without malice, cunning, or regard for consequences. There is no hidden meaning in their behavior, it represents no inferiority compensation or striving for leadership. These children have few friends and seldom belong to gangs, but are avoided and called crazy by their fellows. It seems worthy of comment, considering their emotional instability and pitiful state,

that these children do not develop more defense mechanisms and more efforts at compensation. Several of our Parkinson boys want to be messengers, which may be compensatory for their physical handicap, but none of them want to be bank presidents. The infrequency of the subconscious compensatory mechanism, which is so common and characteristic in psychopaths, seems quite significant.

On admission to the hospital the encephalitis child makes a different impression and reacts in a different way to that of the psychopathic child. The former soon gets acquainted, shows marvelous ability to remember names, and is in close contact with the life about him. But his behavior disorder continues, and he cannot help it; although it may be modified by his being better understood or having better supervision. The psychopathic child, on the other hand, is quiet, reserved and slow in getting acquainted, his behavior disorder may have suddenly disappeared from the hour of admission, but if not it rapidly improves. We usually have a model child, for a time at least, until he goes home. If he remains long he commits some premeditated antisocial act, or escapes by some ingenious plan.

The emotional disorder of chronic encephalitis seems to be the factor of greatest importance from the psychiatric point of view. It deserves careful study, not only in understanding the behavior disorder of encephalitis, but also in affording a new approach to the pathology and mechanism of emotion. Some form of emotional disturbance has appeared to be the most constant clinical factor in the cases studied, through all phases from the restless naughty child to the adult Parkinson with a reactive depression. The emotional disorder seems to be the activating force of much of the behavior. It seems to be the basis of the impulsiveness, and of the characteristic friendly, obtrusive, and often extremely affectionate attitude of these children. Temper outbursts, crying spells, and various manifestations of emotional instability are common clinical features. The explosive outbursts of temper seem significant. While these children react with an excess of emotion when not allowed their own way, there is also a frequent history of severe temper outbreaks which come out of a clear sky and are not temper tantrums with a motive. These outbursts seem more comparable to those observed in traumatic and other organic conditions. Some

patients can feel these attacks coming on, and one girl asks the nurse or teacher to hold her to prevent injuring others. In several ways the emotional factor seems fairly characteristic, and apparently accounts for some of the outstanding clinical features of the behavior disorder.

In addition to the more characteristic features outlined above, some of these patients present other features of a more distinctly psychopathic type. These include sexual perversions, erotic tendencies and delinquency, criminal traits, hebephrenia, and mental deficiency. In most of these cases evidence has been present which would seem to justify their being explained on a constitutional basis, although possibly activated by encephalitis. These cases make up the borderline, and accurate diagnosis is sometimes difficult.

The various ways in which the behavior disorder of encephalitis may terminate are not yet clear, although we have been able to follow the course of many cases over a period of five to ten years. The most common ending is in a slowly progressive and overshadowing Parkinsonism, while apparently the least common ending is complete recovery. The fate of the others remains to be seen. Some will probably go one way or the other in time, but there seems to be a fairly definite group most of whom have left the hospital, but with some degree of emotional instability, behavior disorder or delinquency persisting.

The frequency with which Parkinsonism has developed in these children, and the fact that in many of them the behavior disorder has persisted, would seem to be of considerable significance in the pathological interpretation. It will be seen from Table 2 that 50 per cent of these children now show some degree of Parkinsonism, and that most of these remain in Kings Park or some other mental hospital. In a number of cases a distinct improvement in behavior has been noted with the appearance of Parkinson signs. In others the behavior has been modified by the motor handicap. But in others there has been no definite change in behavior. One Parkinson of three years standing was recently noted as being most of the time disturbed, fighting, and stealing from other patients. In another the behavior is increasingly bad, with vicious impulsive assaults. In a majority of the cases the behavior seems to be modified by the physical state, which overshadows rather than replaces the behavior disorder. The mental and emotional factors are still present, but their expression is hindered by the motor handicap.

The increasing muscular rigidity, masked facies, and slowness of response often convey the impression that the patient is mentally dull when he really is not. The slowness of response is only on the motor side. We can see that he thinks rapidly enough, but his motor expression is delayed. It is especially in patients with involvement of the speech mechanism that we get the impression of a fairly active mind, but with every avenue of expression effectively blocked. In some of these patients there is not only a speech

TABLE 2.

PRESENT STATUS * OF 144 CASES ADMITTED AT KINGS PARK TO
JANUARY 1, 1929.

Acute Phase. Delirium, fever, focal signs, increased cells in sp. fl., etc.—6 cases; discharged recovered—3; died in hosp.—1; remaining in hosp., early Parkinson?—2.

Chronic Phase. "Post-encephalitis" psychosis or behavior disorder.

Age onset acute phase	Total cases	Total Parkinson cases	Now in hospital			Died in hospital		Out of hospital		
			Parkinson	Some signs present	No gross signs	Parkinson	Others	Parkinson	Discharged recovered	Others
4.....	13	3	3	5	0	0	0	0	1	4
5-9.....	58	27	21	14	3	1	2	5	2	10
10-14.....	43	23	14	3	3	2	1	7	4	9
15-19.....	12	9	5	1	0	2	0	2	1	1
20-24.....	6	6	5	0	0	1	0	0	0	0
25-41.....	6	5	4	1	0	0	0	1	0	0
Total.....	138	73	52	24	6	6	3	15	8	24

* In a few cases last known status was used as patient could not be followed.

difficulty, but an actual stopping, the impulses do not come through. The patient gets out a word or two, but the others stick in his throat, and he smiles. The persisting mental alertness is indicated by the fact that some of them read a good part of the time.

Even in well-developed Parkinson patients the characteristic emotional instability, with outbursts of temper, quarrelling and impulsiveness persist, but with a gradual decrease in intensity and frequency. During a period of excitement these otherwise helpless patients show unexpected motor ability. The most helpless patient in the hospital, who cannot stand alone or sit up in bed, has outbursts of temper in which she is destructive of everything in reach, and one night recently she chased another patient to the other end of

the ward. The open attitude and good contact also persist in many cases. The smiling boy with his mouth still open is a distinct type. A progressive dullness seems to be the most common termination of the emotional disturbance. Most of the Parkinson patients who have left the hospital and have been followed at the clinic have been noted as showing emotional dullness, slumping, or deterioration. In some cases, however, transient but rather severe reactive depressions have occurred as the patients grew older. This is the most common type of emotional reaction in adult Parkinson patients.

It seems, then, that the behavior and emotional disorders run their course along with but rather independently of the evolution of Parkinsonism, and that the latter cannot be considered to replace or be complimentary to the behavior disorder.

The relation between the behavior and emotional disorders on the one hand and the neurological signs and organic pathology on the other is of great importance, and correspondingly difficult. The impression gained from this study is that while no very close correlation is to be made between the behavior disorder and the neurological signs, yet the behavior disorder does seem to be associated with the presence of a persistent organic process as reflected in the neurological signs. At first the behavior disorder and persisting neurological signs were looked upon as after effects or residuals of encephalitis in children, while Parkinsonism was considered the chief residual in adults. There has since developed, however, good evidence and opinion that the late neurological manifestations of encephalitis are the expression of a persisting inflammatory process in the nervous system. It is in some respects comparable to that in paresis. It now seems very well established that Parkinsonism is due to destruction of the substantia nigra, by a process which McAlpine describes as "subacute inflammatory changes."

That the occurrence and the clinical course of the behavior disorders in children are to be associated with a persisting active process in the brain is indicated by the following evidence in this series of cases. In the first place, the occurrence of the behavior disorder showed a close relation to the onset of encephalitis in practically all cases. Every case except one had a history of a definite attack or onset of encephalitis at a definite time. Nearly all cases had a history of a typical febrile onset with delirium or marked lethargy which left no doubt of encephalitis. Many of the children had been in bed for weeks or months, and quite a number had been

in hospitals. In very few cases could the acute attack be classed as mild or ambulatory, and there was little opportunity for confusing it with influenza. In six cases there was a history of a definite onset of neurological manifestations without a history of a previous acute febrile attack. In four of these the onset was marked by the appearance of spasmodic twitchings, and in two by typical respiratory disorders. In each of these six cases the behavior disorder appeared with or soon after the appearance of these neurological disturbances. In most cases the time of onset was stated by month and year, and in some the exact day was given. In others the onset was given as occurring at a certain age or in a certain year. With a history of a definite onset at a definite time it was possible to arrange Table 2 according to the age of onset of encephalitis.

Behavior and emotional changes followed the acute phase or onset without a definite interval of complete recovery in all but eight cases, or 7 per cent of the 114 cases in which the onset occurred before 15 years of age. The frequency with which the acute delirious or lethargic onset in children merged into or was followed immediately by emotional irritability, impulsiveness, and a restless overactivity indicates that the beginning of the behavior disorder was a further manifestation of a still active process rather than of a secondary or residual nature. In the eight cases the interval varied from a few months to one year. Of the 24 cases with onset after 14 only three had a history of an interval of complete recovery between the onset and the later behavior disorder or Parkinsonism with psychotic reaction. In this interval most patients were described as changed, nervous, and easily fatigued.

Not only was the occurrence of the behavior disorder closely related to the onset of encephalitis, but also the persistence of the behavior disorder was associated in a large majority of cases with persisting organic signs. Nearly all of these patients were first admitted to a mental hospital two to five years after the onset of encephalitis. On admission most cases showed some neurological signs, such as cranial nerve palsies, tremors, tics, respiratory disorder, or persisting sleepiness, while in some cases definite Parkinsonism was already apparent. To what extent the various neurological signs to be found in patients with a history of encephalitis, and before the appearance of Parkinsonism, are to be considered as residuals due to damage at the time of the acute onset of the

disease or as evidence of a persisting chronic inflammatory process requires further detailed study. The neurological picture seems to be a changing one, and a set systematic examination by the same observer should be made at regular intervals from the onset in order to obtain the proper data. The prognostic significance of various signs could then be worked out. Some signs seem to be more indicative of an on-coming Parkinsonism than others.

The most definite evidence of a persisting brain process in these behavior cases, in view of the later work in pathology, is that approximately 50 per cent of these children now show varying degrees of Parkinsonism. In many of these it has been definitely and in some rapidly progressive, so that extreme degrees of disability have been reached. As indicated in Table 2, the appearance of Parkinson signs has shown little regard for the age of onset. Neither have they appeared at any definite time after onset. In some cases tremors, rigidity, one-sided facial weakness and other signs have been present soon after the acute attack and have progressed to well-developed Parkinsonism. In most of the cases, however, more definite Parkinson signs, such as the masked facies, muscular stiffness, speech difficulties, changes in gait and in muscle tone and position of the arms, have not developed until months or years after the acute onset. This appearance of Parkinsonism in children at varying intervals after the acute attack, up to five or six years, corresponds with the occurrence in adults. The difference is that in children the interval is fully occupied with the behavior disorder while the apparent period of recovery in adults has been frequently reported. It seems significant that adults during this interval do not show psychopathic traits to a noticeable degree.

In those patients who have thus far failed to show definite Parkinson signs some further time and study will be necessary toward the correlation of the course of the behavior disorder with the organic features. It seems, however, that with improvement of the behavior disorder there is a definite tendency for the neurological signs to disappear. This would indicate a cessation of the organic process. Of the eight patients discharged as recovered, none showed definite signs at the time of discharge. In these eight patients neurological signs were never prominent, although there was no doubt as to the diagnosis except in one case, in which there was considerable evidence of psychopathy. In five of the eight,

however, a definite and persistent respiratory disorder was present, in each of which it cleared up as the behavior improved.

It has been noted in a number of cases that a close relation seemed to exist between the respiratory disorder and the emotional factor, and that as the respiratory disorder improved the behavior improved. If we look upon the respiratory disorder as organic this is further evidence that the behavior improves as the organic process subsides. There is good reason to think that the respiratory disorder is due to involvement of the respiratory mechanism in the process going on in the brain. And this does not eliminate the fact that respiratory attacks often are brought on by external emotional situations.

The six patients who remain in the hospital without signs are fairly well accounted for. One girl has been returned because of her persisting erotic behavior; another shows many features of hebephrenia and her peculiar gait has been classed as a mannerism; another girl has been returned because of persisting emotional instability which led her to run away from a bad home situation; one boy retains a very bad temper but is otherwise much improved after five years in the hospital; one girl with a history of typical onset and severe behavior disorder, but never with prominent neurological signs, has gradually improved after five years in hospitals; and one boy continues to show emotional instability, which causes him to fight easily, and a respiratory disorder which is still present seven years after the acute onset.

Discussion.—From this study the impression has been gained that the occurrence and clinical course of the behavior disorder in children after encephalitis can be correlated with a persisting process in the brain. If true it is only a step, of course. How it works is the thing we want to know. Various theories have been advanced. Auden brings in the theory of Head and Rivers of two stages in the evolution of the nervous system and implies that encephalitis has some selectivity of this kind in its involvement of the nervous system. The distribution of the lesions however are primarily vascular rather than phylogenetic. We do not know why certain diseases and certain toxins select certain parts of the nervous system.

Hill discusses several theories and offers one of his own, that involvement of the thalamus causes a release of primitive and in-

instinctive tendencies with an increase in their affective accompaniment. We can agree with him to the extent that the emotional factor seems to be the key to the situation. But whether it is due to a specific lesion of the thalamus remains to be demonstrated. There is, of course, a deep seated opinion with neurologists that "the thalamus is the seat of the affective life," and encephalitis seems to offer an excellent case material with which to prove it.

It does not seem that the behavior disorder, which is much more emotional than motor, is to be directly correlated with the disturbance in the extrapyramidal motor apparatus. There may be a specific area in the thalamus or elsewhere, involvement of which disturbs the emotional function. It is true that not all cases of encephalitis develop mental disorder, and they may not have the same distribution of the lesion. The late appearance of Parkinsonism in children who have not shown emotional or behavior disturbances in the interval would be significant.

In considering the question of a specific localization due recognition must be given to the occurrence of behavior disorders in children from other organic and toxic causes. Twenty-seven years ago Still described cases of "loss of moral control" in children with brain tumor, and after brain trauma and various acute infectious diseases. The behavior disorder and emotional instability were very similar to what we see in encephalitis. Since then cases of this type have been frequently recognized. Strecker and Ebaugh reported a series of 30 cases after cerebral trauma and pointed out their resemblance to encephalitis cases. It seems, therefore, that the mere presence of a brain lesion may be sufficient, when other factors are present. The matter of age is one factor, for these behavior disorders seem to be clearly confined to childhood and adolescence.

As for a possible relationship between behavior disorders in which encephalitis or some other organic lesion is a determining factor and those of a constitutional psychopathic nature, it would seem that the most probable common factor lies in the function of emotion. The emotional dysfunction in the organic cases may not be of the same nature as in the constitutional cases, or it may be more severe, a matter of degree. It is in the later phases of the behavior disorder of encephalitis, where the organic process seems to be clearing up, but with some emotional instability persisting, that the behavior picture and reaction show more similarity to those of

the constitutional psychopath. The same may be true of cases in which the organic process and the behavior disorder have been less severe than in most cases of this series. The late appearance of Parkinsonism in the milder cases, and in those whose behavior disorder has been more distinctly of a constitutional psychopathic type with psychogenic features, but with little or no evidence of an active organic process as indicated by persisting neurological signs and with a doubtful history of encephalitis, probably would be the most convincing proof that the behavior disorder had been actually determined by encephalitis. Such cases would be of the greatest value in indicating a relationship between organic pathology and behavior disturbances of functional and emotional nature.

Treatment of clear-cut cases of chronic epidemic encephalitis on the basis of a persisting inflammatory process of infectious nature demands serious consideration. Poor results from malaria have been reported by some observers. Any suggestions as to treatment would be welcomed.

CASE I.—C. G. Admitted March 8, 1926. Age 15. Female.

Family History.—Father had bad temper outbursts. One sister died of poliomyelitis.

Past History.—Born October 18, 1910. Had been a happy, sociable and affectionate child.

Onset Encephalitis.—October, 1918, age 8. Lethargic type. Slept most of time, difficult to rouse. Continued in a dazed and dozey state for one year.

Behavior Disorder.—Gradually became idle, mischievous, spiteful, and poor in school. After 1922 was worse, contrary, noisy, and impulsive.

Onset Parkinsonism.—October, 1924, age 14, six years after onset. She failed physically, lost weight, hands shook, mouth dropped open. Bad behavior continued, restless and impulsively assaultive. Was difficult for family to control. Because of violent fits of temper and impulsive fighting was sent to hospital in March, 1926.

On Admission.—Parkinson attitude and facies, drools saliva, speech slow and drawling, marked tremors. (Parkinsonism had evidently developed rapidly in 18 months since October, 1924.) Was dull, untidy, stubborn, had a bad temper and was in frequent quarrels. In December, 1926, was unsteady on feet.

1927.—Neurological signs progressive, weak and unsteady on feet, later falling backward. Speech defect more marked. Quarrelsome.

1928.—Troublesome and in frequent quarrels. Progressively weaker, in bed and spoon-fed.

March, 1929.—Up and about. Still in frequent quarrels. Alert and in close contact. Says, "Hello doctor." Masked facies, mouth open, speech defect marked, tongue to left and tremulous. Tremors of fingers. Right arm hangs dead as she walks.

CASE 2.—S. P. Admitted August 4, 1924. Age 10. Female.

Past History.—Negative. Bright and active until onset.

Onset Encephalitis.—1919, age 5. In bed several weeks. Diplopia, ptosis, and facial paralysis.

Behavior Disorder.—After this became irritable, impulsive and assaultive. For past two years has slept more in day and has been restless and complaining at night. Truancy, stealing. On street late at night. Sent to hospital after neighbors complained.

On Admission.—Pupils unequal and sluggish. At first restless, impulsive, abusive, obscene and assaultive. Behavior improved rapidly and was paroled home November, 1924.

May, 1925.—Returned to hospital. Did very well first few months, but home situation was bad, and she again began fighting, stealing, impudent and truant.

October, 1925.—Has been very well behaved in hospital. Paroled.

May, 1926.—Returned. At home was soon fighting, begging money, out late at night, truant, steals money.

February, 1927.—Did very well in hospital, but lazy and had some fits of temper. Discharged.

July, 1927.—Readmitted. Had been noisy and troublesome at home. Led younger children into mischief. Stole money for candy. In hospital was sullen, stubborn, impudent. Right knee-jerk diminished.

1928.—Behavior gradually improved. Sullen at times.

April, 1929.—Sulking, teases other children, petty stealing. Slight left facial weakness. Left arm carried in half flexed position. Left leg slightly stiff, jerking of foot in walking. Nurse says, "She jerks her left side when she walks if no one is watching her." Left knee-jerk not elicited, right diminished.

NOTE.—Apparently beginning Parkinsonism nearly 10 years after onset of encephalitis. During the interval behavior disorder persisted without definite neurological signs being noted. An emotional instability was most definite factor present during this period.

CASE 3.—C. H. Admitted November 12, 1924. Age 17. Female.

Past History.—Bright, active in athletics, good disposition. One year high school.

Onset Encephalitis.—March, 1922. Age 14. Lethargic type. In bed three weeks. Continued dazed and doxy for several months. Would go to sleep while eating or talking. Returned to school after two months but was too doxy and tired to study.

Behavior Disorder.—Became nervous, snapping fingers and kicking, made peculiar sounds and had attacks of dyspnea. Would become rigid, raise arms and fall to floor. In April, 1924, menses stopped and attacks became more severe. Became more irritable, excited, attacks of screaming, cried easily, lost interest in books, slow in dressing, restless at night.

On Admission.—Restless and excitable, talkative and flippant. Respiratory attacks, occurring usually in presence of physician. Frequent masturbation. Erotic, which seemed to be associated with respiratory attacks and emotional excitement. Tremor of facial muscles.

January, 1925.—Very well behaved and doing school work. Masturbates. In a respiratory attack fell and struck face on radiator.

April, 1925.—Trying to control masturbation. Improving.

May, 1925.—No more dyspnea. No masturbation noted. Doing good work in class. In good contact.

July, 1925.—Improved in every way, agreeable and happy.

August 2, 1925.—Paroled. Apparently recovered.

May, 1926.—In business school. No neurological signs, no emotional or conduct disorder.

August 2, 1926.—Discharged. Recovered.

January, 1929.—Mother writes is entirely well, no emotional instability, working steadily.

NOTE.—Respiratory disorder, behavior disorder, emotional instability, and masturbation all improved at same time.

BIBLIOGRAPHY.

- Auden, G. A.: Encephalitis Lethargica. Psychological Implications. *Jr. Mental Sc.*, 71: 647, October, 1925.
- Beverly, B. I., and Sherman, M.: Post-Encephalitis Behavior Disturbance without Physical Signs. *Am. Jr. Dis. Children*, 27: 565, 1924.
- Bond, E. D., and Partridge, G. E.: Post-Encephalitic Behavior Disorder in Boys and Their Management in a Hospital. *Am. J. Psychiat.*, 6: 25, July, 1926.
- Freeman, W.: Specific and Non-Specific Remedies in the Treatment of Encephalitis. *Jr. Am. Med. Assn.*, 89: 1317, October 15, 1927.
- Greenfield, J. G.: The Pathology of Epidemic Encephalitis. *Jr. Mental Sc.*, 73: 575, October, 1927.
- Hill, T. R.: The Problem of Juvenile Behavior Disorders in Chronic Encephalitis. *Jr. Neurol. and Psychopath.*, 9: 1, July, 1928.
- Kirby, G. H., and Davis, T. K.: Psychiatric Aspects of Epidemic Encephalitis. *Arch. Neurol. and Psy.*, 3: 491, 1921.
- Marshall, R. M.: The Mental Aspects of Epidemic Encephalitis. *Jr. Mental Sc.*, 73: 589, October, 1927.
- MacKenzie, Ivy.: Epidemic Encephalitis. *Jr. Mental Sc.*, 73: 567, October, 1927. Also *Br. M. Jr.*, 1927, 2: 532.
- McAlpine, D.: Anatomic-Pathological Basis of Parkinson Syndrome Following Epidemic Encephalitis. *Brain*, 49: 525, December, 1926.
- McCowan, P. K., and Cook, L. C.: Chronic Epidemic Encephalitis. Treatment by Malaria. *Lancet*, II, 1927, p. 861. See also Editorial, p. 873, and Craig, R. N., p. 860.

- Parsons, A. C.: Report of an Inquiry into the After-Histories of Persons Attacked by Encephalitis Lethargica. Report No. 49. Ministry of Health, London, 1928.
- Still, G. F.: Some Abnormal Psychical Conditions in Children. *Lancet*, 1: 1902, 1008, 1077, 1163.
- Strecker, E. A., and Ebaugh, F. G.: Neuropsychiatric Sequelae of Cerebral Trauma in Children. *Arch. Neurol. and Psychiat.*, 12: 443, October, 1924.
- Wimmer, August: Chronic Epidemic Encephalitis. Wm. Heinemann, London, 1924.

DISCUSSION.

DR. EARL D. BOND (Philadelphia, Pa.).—This experiment that has been going on at the Kings Park Hospital with these children has been of tremendous interest to us at the Pennsylvania Hospital who have been carrying on a similar experiment for about the same time. When I looked over the abstract of this paper, however, I was rather disappointed and thought that I might have to revise my opinion as to the prognosis of these children in a downward direction. After hearing the full paper, however, I think that the experience of the Kings Park Hospital and the Pennsylvania Hospital is really a great deal better than it seems to anyone who has considered the question once.

In the first place, the Pennsylvania Hospital has ruled out cases of Parkinsonian syndrome and has ruled them out because it thought there was no hope. Then it has ruled out all children over 12. The Kings Park experiment has been broader than our own, but it is really two experiments in one. If you take the Kings Park tables, which I had a chance to see before the meeting, and rule out the Parkinsonian syndrome and then rule out the cases over 12, you come down to about 50 cases, which show no continuing neurological disorder and are under the age of 12. Dr. Gibbs has reported, seven recoveries in that group of 50. Our own experience gives like results.

In addition to that, we now have in the wards 25 cases who are being kept there not because they are going badly, but because they are going so well that we want to continue treatment for another six months, and we feel that the present group is going to do better than the others.

Concerning the bright children who are badly behaved, who have no continuing lesion, I should agree with all of Dr. Gibbs' positive statements. There was one negative, however, that I cannot agree with. He says there is no depth to the inner mental life, no reaction to inferiority feelings. Our children are full of a vivid and intense mental life and full of all sorts of reactions to inferiority feelings. Dr. Partridge, whom I see here, has some remarkable examples that he can give among our children of the classical reaction to feelings of inferiority.

Of course, there is much to be said about this subject in all ways. The last thing I want to call to your attention is to emphasize what Dr. Gibbs says, that this is a bridge to a consideration of bad behavior everywhere and that behavior disturbance in the bright children who have not Parkinson's disease is a continuing opportunity for psychiatrists.

DR. EDWARD A. STRECKER (Philadelphia, Pa.).—Dr. Gibbs has presented a very valuable and interesting summary of the late results in this problem. I would simply like to say that since we cannot possibly hope to know to what degree the behavior of these children may be influenced by treatment and since we lack any specific method of approach, our best chance at the present time is along the lines of long-continued and standardized training. The behavior disorder ought to be considered first in itself, that is, in so far as it may be related to brain damage or to continued irritative inflammatory reactions in the brain, and, second, in so far as it may be conditioned by additions to the behavior due to the lack of understanding on the part of the family and the environment in which the child is placed at the beginning of his behavior disorder.

There are, of course, very serious errors, both in the direction of omission and commission which complicate the behavior to a very great degree.

As to the treatment itself, it would seem fair to consider five points: First, that the child be taken away from his environment as soon as possible; second, that the treatment be instituted early; third, that the treatment be continuous and for a long period of time; fourth, that the keynote of the treatment be an impersonal attitude, that the behavior disorders of the child be met by an impersonal attitude on the part of those who have the responsibility for the treatment of the child; fifth, that that attitude be not only impersonal, but that it be constant in its impersonality and continuous and similar with the hope of establishing more favorably conditioned reflexes of behavior; and, finally, that following the discharge of the child, there be a follow-up and a gradual stepping back to a more useful condition of life.

In regard to the Parkinsonian cases, my own experience has been only with those cases in which the Parkinsonism followed as a continuation of the acute phases of the illness and in those cases the behavior disorders were either absent or of no significance.

DR. MORGAN B. HODSKINS (Palmer, Mass.).—I am associated with a hospital for epileptics and we have had several lethargic encephalitis cases admitted to the hospital who had convulsions. Dr. Gibbs did not say whether any of those children he described had convulsions or not. The interesting point in that is that they also have their behavior problems and that as Parkinsonism develops, the convulsions decrease in number. We have a few in the hospital at the present time whose convulsions have entirely ceased, which is along the same line as we found in deterioration in epileptics. In epileptics, as extrapyramidal symptoms develop, we found that the convulsions decreased in number.

DR. CHARLES E. GIBBS (Kings Park, N. Y.) in closing.—First, to answer the question about convulsions; they have occurred in very few of our patients. One girl after several years of severe behavior disorder and extreme overactivity died after a series of convulsions without having had con-

vulsions before. In another patient convulsions occurred for a short time, which seemed to mark the transition from the period of behavior disorder to the beginning of the Parkinson phase.

DR. HODSKINS.—Did the convulsions decrease in number?

DR. GIBBS.—They only had a few.

With regard to the mental content and defense mechanisms, I think the difference in our observations and those of Dr. Bond's may be accounted for in the selection of cases. Our cases, of course, were primarily severe behavior disorders and were not selected by us but were committed patients and so represent the more severe type. Probably his patients correspond more to those in our series who have improved and left the hospital or remain without very severe behavior disorder and in that respect approach the psychopathic children. It is in this group, who seem to be gradually improving but with some residual behavior disorder or emotional instability, that we may have some connecting link between encephalitis and the abnormal behavior seen in psychopathic children.

MIGRAINE EQUIVALENT.

By JOHANNES M. NIELSEN, M. D.,

Associate in Neurology, Battle Creek Sanitarium, Battle Creek, Mich.

Psychic disturbances entirely replacing the usual symptoms of an attack of migraine have led to a great deal of discussion and some controversy during the last three decades. All students of the question recognize that mental symptoms occur in migraine, and that they may precede, usher in, be concomitant with, terminate, or follow an attack. Clinicians experienced in migraine also recognize that mental symptoms may occur in migrainous patients apart from and between the episodes usually called attacks of migraine, but authorities are divided on the question of whether or not these mental symptoms are migrainous in nature.

The reason for the controversy is that the mental disturbances usually encountered resemble closely both epilepsy and hysteria, and migraine is not only closely allied to the former but actually considered by some a manifestation of epilepsy.

Tissot is quoted by Moersch¹ as having recognized psychic equivalents of migraine as early as 1834. Liveing² believed such symptoms to be an expression of migraine, and Mingazzini was so certain of this that he suggested the establishment of a separate category "dysphrenia hemicranica" for these manifestations.

Krafft Ebing³ considered such attacks epileptic and Oppenheim⁴ still stated in 1911 that "it is doubtful whether mental disturbances occur as equivalents of hemicrania although such disorders appearing in those suffering from migraine and apparently replacing the paroxysm of pain have been interpreted in that way."

Edward Flatau⁵ summarizes the question well. He believes such attacks migrainous, but states that the term migraine equivalent should not be used unless: (1) The psychosis occurs in a person who also suffers from unquestioned migraine attacks; (2) when the cessation of the hemicrania and the beginning of the psychosis approximately correspond in point of time; and (3) when the form of the psychosis is the form most frequently encountered in

migraine, *i. e.*, confusional states. This author gives 15 pages of references on migraine.

Moersch¹ in an excellent paper on Psychic Manifestations in Migraine expressed his belief in the migrainous character of such attacks and states, "It is possible that many of the peculiarities which are ascribed to certain persons, such as occasional vague, visual phenomena, mild recurring headaches, and apathy, periodic mental dulness, or other periodic mental fluctuations, are larvated forms of migraine." But White² in his Outline of Psychiatry does not mention migraine equivalents and hence we can assume that he classifies such disturbances under some other head.

In presenting these statements, no attempt has been made to include the opinions of all writers on the subject but merely to show how recognized neurologists have differed.

The writer has encountered a strikingly clear case of what some authors class as migraine equivalent and since cases so pure as this are obviously exceedingly rare, a report is thought desirable. The case also shows that the term "confusion" is not always sufficiently specific.

The patient is a physician, aged 37, who comes with no complaint except episodes of mental disturbance. These do not incapacitate him; but he has worried for years about their possible significance, because genius and dementia præcox occur in the family of a paternal half uncle; and he has consequently feared for his own mental state. Aside from the above facts he knows of no nervous or mental disturbances or of migraine in the family.

Physically the patient appears very healthy. He is 67 inches tall, slightly obese, weighing 170 pounds. Osseous, muscular and cutaneous systems show nothing of note. He has suffered since childhood with marked dental decay, repeated attacks of pansinusitis and with a chorioretinitis, probably due to the latter. There has been no evidence of activity in the chorioretinitis for 28 years. The macula has been destroyed in the left eye and a false one has developed, leading to a moderate strabismus. Heart and lungs show no pathology, but the blood pressure is 94 systolic, 66 diastolic. The arteries are soft. All deep reflexes are active but within normal limits. All the usual laboratory tests on blood, urine, etc., are negative.

The attacks of mental disturbance may be described as follows: He feels a mild emotional depression come on. Things in general seem serious. Then a feeling of strangeness supervenes and this is followed by a peculiar confusion.* During this he is perfectly oriented yet cannot give an orderly

* The word confusion is not here used in its psychiatric sense to mean complete disorientation in all spheres, but in its ordinary sense.

account of anything. He is in possession of a multitude of facts concerning medicine and his environment, but there are large defects in his memory and hence organization of his knowledge is lacking. Actually the defects are so large that it seems to him as though only small groups of facts make their unbidden appearance in his consciousness. This isolation of facts and events, he says, is the outstanding difficulty in the episode. There is no association of any kind between these groups of facts. Further, he remembers nothing during an attack for more than half a minute. He says he feels as though he sees events through a long tube or under the high power of a microscope and is unable to establish the relations between the various fields observed. He can move the tube to another field, but then he has no recollection of the one just observed. This soon leads to confusion.

Practically, this state has the following effect. He cannot recall events, lodge pass-words, formulas, dates, names, appointments, or promises at will. They may all come to him but he cannot command them. It is difficult for him to carry on a conversation because he cannot recall at any moment the last statement. By the time he receives a reply to a question he does not know what he has asked. Consequently he cannot deal with people, cannot take a history from a patient, cannot dictate. He repeats questions and appears to others very absent-minded. He feels as though he were in unfamiliar surroundings and everything seems unreal. The unreality is the most distressing, feature of the whole episode, he states. Fortunately the disturbance lasts only from three to six hours and the episodes appear only about once in every 6 to 12 months. After the episode he has a perfect recollection of the events and can recall all his confusion and embarrassment.

The early history of these attacks is difficult to obtain. He recalls vaguely such disturbances in his childhood, but an attack at the age of 18 has left a lasting impression. He was about to go to a lodge meeting when it appeared. He moved as in a daze, mailed a package with only a return address on the envelope. He forgot his dues book and the pass-word and it seemed that he was moving in a strange world. He did not avoid traffic on the street and was warned by a policeman to watch where he was going. He reached the hall early and met a friend who gave him the pass-word. He entered the meeting in a distressing confusion, took no part in the discussion but sat quietly for about an hour when the attack wore off; and by the time the meeting was over his mind was clear. He developed an aversion for the attacks and wondered whether he would lose his mind.

After studying medicine he became more observant and noticed that a severe nervous or mental strain frequently preceded the attack. But not by any means did every severe strain bring on an episode. The disturbance still occurred only once or twice in a year.

He was questioned for symptoms of migraine but persistently denied any tendency toward migraine until fractional attacks were discussed and fortification spectra were shown to him. It was then revealed that he suffered from attacks which he had never understood, referable only to the eyes. These attacks also followed unusual strain but were never associated with the mental

symptoms described above. They began usually as blind spots with concentric rings turning in the upper right quadrants of the fields of vision. Soon they would spread to become a quadrant amblyopia or a right homonymous hemianopsia. At times both upper halves of the fields of vision were involved. He had great difficulty in driving a car during an attack and could not read ordinary print because of inability to follow a line. These disturbances were always sooner or later accompanied by typical fortification spectra. These attacks were never accompanied by nausea, headache, pain, dizziness or weakness, but were associated with a very mild confusion. They invariably lasted only 15 minutes to one hour and were considerably more frequent than the attacks of confusion described in the foregoing.

The patient obviously had migraine but what were the attacks of confusion? Careful analysis showed that amnesia of a peculiar type was all the mental pathology present. The patient's statements that he suffered from isolation of facts, lack of association, facts coming out of the past without association or connection, inability to carry a chain of thought or to concentrate, unreality, and the specific complaint of a very short memory—all indicate that amnesia is the basic defect. It was associated with a depression. To call the mental state confusion does not aid in reaching an accurate analysis. In fact, this is a defect in many records. Here we have the advantage of a patient educated in medicine and able to give valuable help in analyzing the confusion.

The patient was seen in one of these attacks. He appeared flighty and not like the composed, sedate man he usually was. He was slightly depressed but also a little irritable. (He was aware of this, spoke of it and explained that he was disgusted with his inability to carry on as well as usual.) Motor activity was normal for the patient. Attention and comprehension were undisturbed but his stream of thought was obviously defective. His power of retention was nearly absent. He ordered a patient undressed and promptly forgot all about him. He called each patient in by name, but had no idea what the status of the case was until records were consulted. During conversation he often forgot the patient's name. Content of thought was normal. Orientation was clear for time, place and person but was obviously defective as to details. Insight was perfect.

He was asked to sit down and write his thoughts about his own condition. The notes were brief, to the point, showed intelligence, insight and keen powers of observation, but a whole page was covered with repetitions of a few statements. He managed to remain on duty in his office, examine patients and prescribe until the usual closing time. Two hours later he was normal.

The difficulty in the diagnosis lay in the possibility that the patient might be suffering from simultaneous migraine and epilepsy, or migraine and hysteria. With reference to the former, there never were any periods of unconsciousness, even in the state described above. He had no petit mal or anything simulating it. He lacked the emotional poverty, the egocentricity, the sadism, in short the epileptic personality. (Doubt has recently been cast on the existence

of an epileptic personality.'') Similarly he lacked the hysterical personality. Flight, actual or symbolic, was distinctly foreign to his nature, in fact he was rather aggressive and preferred to invade the enemy's camp. I therefore considered the attacks in question migrainous—mental equivalents of migraine.

Schroder * has called attention to the medico-legal significance of the condition and has presented an excellent report with careful analysis of a case. However, episodes of mental disturbance were associated with obvious signs of migraine, and the patient was continuously abnormal in his behavior even when not suffering with an attack.

The writer does not wish to predicate of migraine all psychic or emotional abnormalities occurring in the migrainous. Nor can he entirely agree with the postulates of Flatau already mentioned. He would prefer to postulate that a migraine equivalent must be a psychic disturbance of short duration (hours to possibly two days) not associated with loss of consciousness, which does not in years become progressively more severe or cause mental deterioration; and it must occur in a patient with first, obvious migraine attacks; second, freedom from an epileptic or hysterical personality make-up and hysterical episodes (flight from reality); and third, an entirely normal emotional and psychic state between attacks.

BIBLIOGRAPHY.

1. Moersch, F. P.: *Psychic Manifestations in Migraine*. Amer. J. Psychiat., 3: 697-716, April, 1924.
2. Liveing, E.: *On Megrin, Sick-Headache and Some Allied Disorders*. (A Contribution to the Pathology of Nerve-Storms.) N. and A. Churchill, London, 1873, pp. 222-223.
3. Krafft-Ebing: *Über Hemikranie und deren Beziehungen zur Epilepsie und Hysterie*. Arbeiten a. d. Gesamt-gebiete D. Psych. u. Neurol., 1897, I Heft, Leipzig.
4. Oppenheim, H.: *Hemicrania (Migraine)*. Text Book of Nervous Diseases, Vol. II, 1911, pp. 1180-1192. Trans. by Alex. Bruce.
5. Flatau, E.: *Die Migräne und ihre Abarten*. Handbuch Der Neurologie, Vol. V., Spezielle Neurologie IV, M. Lewandowsky (Julius Springer, Berlin), 1914, pp. 342-411.
6. White, Wm. A.: *Outlines of Psychiatry*. Nervous and Mental Disease Publishing Co.
7. Notkin, J.: *Is There an Epileptic Personality Make-Up?* Arch. Neurol. & Psychiat., 20: 799, October, 1928.
8. Schroder, G. E.: *Hemicrania Psychica, Taagetilstand*. Ugeskrift for Laeger., 82: 1103, August 26, 1920.

THE RÔLE OF TRAUMA IN VARIOUS NEURO-PSYCHIATRIC CONDITIONS.*

By MICHAEL OSNATO, M. D.,

Director, Department of Neurology, New York Post-Graduate Medical School and Hospital.

By far the most harassing and certainly the most frequent group of cases which develop neuropsychiatric symptoms following injury are the cases of so-called post traumatic neurosis. I have come to limit this term exclusively to patients who have reacted emotionally in a direct way to injuries, often quite trivial in themselves and not generally associated with definite evidence of organic brain or other central nervous system disturbances. As I pointed out some years ago in several articles published in the *Neurological Bulletin of Columbia University*,^{1, 2} this type of case should be separated from the cases showing somatic conversion hysterical phenomena. In the cases of traumatic neurosis I find that the essential feature motivating the emotional reaction is the direct reaction of the suddenly tremendously disturbed ego to the element of terror and physical discomfort associated with the injury. The psychologic explanation of the continuing symptoms in these cases follows the Lange-James conceptions, further elaborated later by William MacDougall,³ and the psychologic principle of the conditioned reflex as conceived by Pavlov. According to these conceptions many things may either consciously or subconsciously reactivate by association the visceral terror and fear reactions caused by the injury and in that way continue to add to the sum of the emotional reactions which are responsible for the symptoms. The emotional tone of consciousness is influenced in such a way by the injury and attendant circumstances that the visceral changes which occur at the time may continue as an emotional expression and may be activated each time that the instinct of fear or horror is aroused long after the injury itself ceases to have a prominent place in consciousness.

* Read at the eighty-fifth annual meeting of The American Psychiatric Association at Atlanta, Ga., May 14, 15, 16, 17, 1929.

With individuals possessing adequate, well-rounded personalities, these emotional reactions do not generally continue for very long. However, they may last until the stimulus arousing the responses is removed. If, for instance, an iron worker has fallen several times from a great height, and his conscious and subconscious reactions to returning to work under circumstances which might make for a repetition of the accident become too disturbing to be controlled, then in exceptional cases, as part of the readjustment of the individual, it becomes necessary to advise a change of employment. These psychologic problems are very easily understood and perhaps need no further elaboration. I have only to recall the case of a splendid type of young iron worker who fell from a great height and received serious injury to the chest and right leg. Except for a simple scalp wound there was no head injury. He developed a series of subjective visceral discomforts and an anxiety state which recurred every time that he attempted to return to his work so that finally we were forced to advise him to find other employment, and he thereafter made a good adjustment.

One must, however, be very careful not to confuse this type of case with those generally referred to as traumatic hysteria. If one adheres strictly to the teachings of certain modern schools of abnormal psychology, the correctness of the term "traumatic hysteria" is certainly open to question. However, for practical purposes in speaking of cases of functional nervous reactions having objective somatic symptomatology and occurring in individuals applying to the courts for compensation because of injury, it may be advisable to continue to use this term.

Early in my experience with the psychoneuroses connected with trauma I became convinced that the individuals who develop the symptoms grouped under the term "traumatic hysteria" were rather different from those for whom I have reserved the term "traumatic neurosis." The mechanisms are also quite different. In summarizing the results of my experience with the cases of traumatic hysteria, I may say that the reactions are not primarily directly determined by any pathological considerations due to the injury but are chiefly conversion phenomena occurring in maladjusted individuals who are apt to be physically, emotionally, and even intellectually inferior types. In respect of the psychological

mechanism; namely, the conversion of the emotional response to difficulties into symptoms, the mechanism is the same in these traumatic cases as in the hysterias of civil life not occurring in connection with trauma. But it is my belief that the traumatic hysterias do differ in that they develop in individuals who are dull or even defective intellectually and these patients often have associated physical and emotional difficulties not related to the accident. No case purely of traumatic fear neurosis ordinarily presents the combination of symptoms commonly known as hysterical stigmata, such as blindness, deafness, dysbasia, astasia, sensory disturbances, paralyses, or amnesias, all of which are so frequently encountered in the hysterical types. In the article already referred to¹ I described the physical and mental make-up of these patients who react to injury with somatic hysterical phenomena. The mechanisms in these cases are often extremely interesting and complex, but frequently quite elemental. The injury serves merely as a convenient compromise by which the patient attempts to bridge over difficulties of adjustment. In the cases of traumatic neurosis, however, the reaction is a simple, instinctive one—the emotional response of the personality to a suddenly overwhelming blow to the ego. Pain and fear, of course, play an important part, but the sudden obliteration of the ego, I am convinced, is a major factor. The fact that an individual may suddenly be rendered helpless, by forces so tremendously greater than himself, is often the cause of certain very distressing reactions. The following case will illustrate some of the mechanisms at work in these cases of so-called traumatic hysteria:

CASE I.—L. I. G., 37 years of age, wife of a successful executive in a large transportation company, was injured while sunning herself at one of the large watering places, on July 13, 1926. Her description of the accident was rather vague, but from others it was learned that while she was sitting on the beach under a large beach umbrella, a strong wind uprooted the umbrella and the pole struck her on the head. The patient said she was dazed but not too dazed, however, to prevent her from making a very strenuous complaint to the management concerning the incident. When she finished making her complaint, which was accompanied by an excessive emotional response, she fainted as a crowd gathered. She was taken to a hospital. There was no cranial bleeding and no scalp wound. She was only hospitalized for several hours. X-ray of the skull was negative and the injury was quite obviously trivial. However, that evening she began to vomit and continued to be nauseated and vomited for about two weeks. She remained in a semistuporous state and either could not or would not give her husband any clear

account of what had occurred. When she got out of bed she began to lose weight, slept badly, was very nervous, depressed, cried a good deal and seemed moody. She avoided her friends and complained of dreams in which she was being killed, or saw murders, and dreamt also of jumping out of the window. She was taken to Europe by her husband on the advice of a family physician. Her husband said that from the moment she got on the boat until they returned she seemed to be an entirely different person. She enjoyed herself greatly and he thought that she was quite well when they came back. Several weeks after coming home she again began to complain and said that she was tired, could not sleep well, felt nervous and depressed. The husband was quite disturbed about her and when I saw her for the first time on December 3, 1926, he was a distracted individual.

I saw this patient not in connection with the claim for damages, but as a private consultant so that I was able to get all of the points of view and to learn all of the determining factors. So many times it is impossible to get at everything, especially if the examiner is seeing the patient for one of the litigants.

Briefly, the history is as follows:

It would appear that a few days before the injury of July 13, 1926, a friend engaged her in conversation and by innuendo disturbed her considerably. The gist of this conversation was that she had heard some talk that the patient's husband had been seen with another woman. She alleged that several people had seen him, always with this lady, and they had noticed that he was very attentive to her. This woman was a distant relative of the patient who occasionally visited them. After the injury to Mrs. G. this relative installed herself in their home and took charge of the establishment. It seemed to the patient during her illness that there was something going on under the surface between this relative and her husband, yet she was never able to find anything tangible. She worried a great deal about this and was uncomfortable all the time that she was at home recovering. However, when she and her husband left for Europe she felt just as though she had been freed from some horrible pressure. She was quite happy and felt well all the time they were away, but when they got home following the trip to Europe she again became much disturbed. This relative came again to visit them. The patient spied on both of them and followed her husband and her visitor. On two occasions they met; once she saw them get into a taxicab and drive off together. The patient's reactions were now quite clear.

I insisted on the litigation which was pending, and which had been originated by the husband, being quickly disposed of.

The neurological symptoms which this patient showed when she was examined on December 3, 1926, were of the type that one usually classifies as hysterical phenomena. They consisted of a tremor of the hands, attacks of amnesia, and somatic sensory disturbances (corneal anæsthesia, anæsthesia of both lower extremities).

On the physical side the striking features referred to a condition which was clinically quite definitely a hypothyroidism of the myxedematous type. The patient had been gaining weight in the last two years, especially about the torso, and in the last six months she complained of feeling heavy and as if she were weighted down. She had the typical puffy eyelids, especially the lower lids; her scalp was very dry and the hair was coming out in alarming quantities; the skin over her shins and the back of her hands was dry; the nails were brittle, especially the toe nails; there was a typical myxedematous feel of the tissues of the shoulders, and in addition a considerable collection of the same type of subcutaneous tissue in the torso.

We were, therefore, confronted with a situation in which there were at least three factors. First, an inadequate personality which made the patient what her husband described as a lamentably suspicious and jealous individual. Secondly, definite conversion, emotional elements caused by jealousy precipitated by trauma. Thirdly, these reactions expressed necessarily through her nervous system were further influenced by the profound metabolic changes caused by dysfunction of the thyroid gland. These were the important factors motivating this patient's symptoms. The injury acted only as a contributing factor to the psychogenic and physical determinants which caused the acute hysterical symptoms. It served simply as a precipitating factor and not at all as a major one.

This case is quite typical of some of the mechanisms which underlie the compensation neuroses of hysterical type. It is unfair to say that they are of traumatic origin. Trauma simply acts as a precipitating factor and as a bridge by which a compromise is effected between the difficulties under which the patient may be laboring at the time of the injury and the aims toward which the patient is striving. These difficulties are converted into convenient, gainful symptomatology. As I have said before elsewhere, in many of these cases the mechanisms are so close to consciousness that one

has difficulty in making the diagnosis between conscious malingering and true hysteria.

A more simple mechanism is seen in the following case:

This patient was a Russian portrait painter, a refugee who escaped from Russia during the revolutions. He was unable to make a living here and dire necessity forced him to accept work delivering paintings for a store in the lower east side instead of painting portraits which he had hoped to do when he came to America. He also helped to sell these cheap paintings, but his main job was to deliver them.

One day while walking on a crowded street a man who was in great haste bumped into him and the patient fell striking his right shoulder. He was carrying a small, framed painting under his right arm. There was no head injury. He was able to get up immediately, but he alleged that he promptly lost the power of movement of the entire right upper extremity and there was a complete loss of all sensation in the entire extremity. I saw him several months later. X-rays of the shoulder were negative. There were no external evidences of injury. Excepting for a small bruise over the deltoid there never had been any evidence of injury. The claimant had not responded to treatment and when I saw him he had what I considered an hysterical monoplegia affecting the right upper extremity. It was associated with anesthesias of the entire right side of the body, including the cornea. The psychogenic factor was so close to the surface of consciousness as to convince others who saw him of the belief that he was a malingerer. Here again the accident acted simply as a precipitating factor. The man had been thoroughly discouraged and bitterly disappointed because he had to perform menial tasks to make a living. He had hoped when he came here to continue his portrait painting and become successful—not only in his art but socially and economically. He desired to be at least as well off as he had been once in his native country. The bitterness of his disappointment and the inadequacy of his personality and of his abilities to realize these strivings made him use the trivial accident as a means of, at least temporarily, effacing the tragic reality of his situation.

The next group of cases are those which Dr. Giliberti and I have recently described in our article entitled "Post Concussion Neuroses—Traumatic Encephalitis."⁴ We developed the conception of the post concussion phenomena as a traumatic encephalopathy. The conclusions reached on this subject were, that "anatomic and clinical investigations seem to show definitely that our conception of concussion of the brain must be modified. It is no longer possible to say that 'concussion is an essentially transient state which does not comprise any evidence of structural cerebral injury.' Not only is there actual cerebral injury in cases of concussion, but in a few instances complete resolution does not occur and there is a

strong likelihood that secondary degenerative changes develop. When this happens, we have a condition which, clinically at least, resembles some of the reactions seen in encephalitis. We feel, therefore, that the post concussion neuroses should properly be called cases of traumatic encephalitis." Perhaps a more correct term would be traumatic encephalopathy.

I want to emphasize the fact, however, that the number of instances in which complete resolution does not occur are, in my experience, very few indeed. Surely in the vast majority of cases of concussion, recovery with almost complete resolution of the diffuse hemorrhagic process takes place, even granting that this occurs to a varying degree in every case of concussion of the brain. It has not been realized, perhaps, how many people in the general population have suffered concussion without, however, so far as one is able to judge clinically, showing any lasting effects. Taking into consideration the number of children who have had falls with resultant injury to the head and varying degrees of the concussion syndrome, and the great number of boys who have suffered concussion in consequence of injuries while at play, and including also those who in adult life fall from horses, suffer automobile and other accidents and industrial injuries of various kinds, one can form a picture of the enormous number of persons in the general population who have, at some time or other, suffered a concussion of the brain. Nevertheless, permanent disabling clinical phenomena following concussion of the brain, with or without fracture of the skull, are not commonly encountered. This is in line with our experiences with cranio-cerebral war wounds. The number of cases of traumatic insanity or epilepsy, or of cases of tumor of the brain which develop following such injuries, is surprisingly small.^{11, 12} Nevertheless, when the hemorrhagic process has been so marked and so diffusely present as in the brain we studied and in Cassasa's cases,¹³ it cannot be otherwise than that certain mental and somatic disabilities, chiefly the former, will follow, developing apace with the secondary diffuse glial and ganglion-cell degenerations. The degree of this diffuse secondary process determines the nature and degree of the clinical phenomena, with one important reservation. Sooner or later in a great many of these cases, however, instinctive or environmental influences, some of which I have already considered in the foregoing illustrative cases, operate to produce a group of emotional

symptoms which it is difficult not to classify as purely psychogenic in origin. Some of the environmental influences are undoubtedly contributed by interested relatives and representatives of the litigants.

An important consideration in connection with the post concussion neuroses is the fact that one must go very carefully into the history of the injury. This is too frequently lightly passed over. I have time and again listened to referees and judges and others accept the word of attorneys and representatives, or of patients, that the claimants were unconscious for hours, or for indefinite periods, without further investigation or questioning. The most striking symptom of concussion of the brain is unconsciousness. It is important to determine, if possible, whether the patient was actually unconscious or whether he simply had fainted or was giddy and dazed for a moment, or fainted later from fright or the loss of blood. Wherever possible the testimony of the first physician who saw the patient immediately after the accident should be relied upon to determine whether unconsciousness was present. The most important symptoms of concussion of the brain are paralytic in nature. The deep reflexes, especially if the concussion is profound, may be diminished, but usually, however, they are fairly lively. If seen very soon after the concussion the pupils are generally quite small and react poorly to light, later the pupils may be widely dilated. There is a marked general vasomotor disturbance with pallor, cold extremities and sweating, low blood pressure at first, sometimes followed by a rather rapid rise if associated with intracranial bleeding, often a very slow pulse in the beginning, later a rapid, small thready pulse, and in very severe cases occasional disturbance of sphincter control. The symptoms of unconsciousness may be the only guide if there are only lay observations to rely upon. For several days following the concussion and often for much longer periods, the patient shows marked mental and physical retardation, often accompanied by confusion, irritability, drowsiness at first and days later great restlessness, and anxiety at night.

The pathology of concussion was gone into very carefully in our discussion of the post concussion neuroses⁴ and need not be touched upon again here. The conclusions arrived at in that study were based upon a study of 100 clinical cases and six cases with

pathological specimens (brain only), one of them very carefully studied from the neuropathological viewpoint.

The symptoms complained of by these patients who have had concussion of the brain eventually become almost wholly subjective, but the pathological basis underlying the situation is, in my opinion, so definitely established that I no longer look upon these symptoms as being necessarily emotional or as having no organic basis and, therefore, I no longer agree with the conception which most men have that concussion of the brain is necessarily an essentially transient state due to head injury and does not include any evidence of structural cerebral damage.

As a typical example of a case of post concussion neuroses, I shall refer briefly to the case of N. R., 38 years of age, male, who while working on the platform of a subway station under construction fell through an opening intended for the stairway, some 25 feet below. He landed on his feet and pitched forward, striking his head against a concrete base. He was unconscious and was picked up and assisted into an automobile and taken to a physician's office. There was a fracture of the nasal bone, but otherwise no skull injury. When I saw the patient three months after the accident there was still a slight difference in the size of the pupils. There was a moderate diminution of the associated movements of the left upper extremity, and he also exhibited an irregular, coarse tremor, worse in the right hand. At rest the tremor had some of the characteristics of that seen in paralysis agitans. At other times the character of the tremor changed and instead of being fine, fairly rhythmical and of very minimal force it became coarse, irregular and of great force and amplitude. The patient was depressed and had great difficulty in sleeping at night. He talked rather slowly, complained of headache, which he localized to the occiput and the back of the neck, and complained also of nervousness and irritability. A neurological examination was otherwise completely negative. There was no anaesthesia of the cornea or of the pharynx or elsewhere. There were no hysterical visual or auditory disturbances and there was no hysterical paralysis in any of the extremities. I believed the tremor, the diminution of associated movements in the left upper extremity, the retardation, irritability, restlessness and anisocoria were all part of the structural reaction of the brain to the trauma and not primarily of emotional origin. This group of symptoms occurs sufficiently frequently following concussion to establish a syndrome and they enable one to diagnose a post traumatic encephalopathy rather than a psychoneurosis of purely emotional origin.

The duration of symptoms following even moderately severe concussion of the brain is sometimes quite long and the disability may be terminated too soon. Each case must stand by itself and no generalizations as to length of disability are possible.

The occurrence of fracture of the skull or other head injury in syphilitic individuals results often in very perplexing problems. The question of determining what influence, if any, the injury had on an admittedly pre-existing syphilis which was presumably latent, is one which comes up for consideration quite frequently. My attitude has been discussed in an article entitled "Trauma and Other Non-Luetic Influences in Paresis."⁶

The following case may serve as an example :

F. A., 33 years of age, male, ironworker, was injured August 27, 1927, while working on a dry-dock. A galvanized pail containing rivets fell 25 feet and struck him on top of the head. He fell to the ground, was apparently not completely unconscious, for he got up quickly, but he was stunned for five or six minutes. There was no scalp wound and no bleeding from any cranial orifice. There was no hospitalization at any time. Soon after the injury he walked several blocks on the way home and suddenly became weak in his lower extremities and lost consciousness for a brief period. When I saw him on November 30, 1927, he had not yet returned to work. He complained of attacks of vertigo upon exertion or upon change of posture, blurred vision, shock-like pain in the head, weakness, especially in the left leg, and sleeplessness.

Examination disclosed a right pupil that was about 6 mm. and completely immobile; the left pupil was about 3 mm., irregularly oval, reacts slightly to light, but did not react to accommodation or convergence. The left palpebral fissure was larger than the right. There was a mild left facial residual weakness. The left knee-jerk was definitely more active as was also the left ankle-jerk. There was a moderate Romberg. The rest of the examination was negative.

We hospitalized the patient. A fracture of the vault of the skull involving the anterior half of the left parietal bone was discovered. The fissure fracture was about 9 cm. long. The blood and spinal fluid Wassermann's were four plus. There were no signs of blood in the spinal fluid. After much questioning we finally obtained the history that he had a chancre 18 years ago. Investigation disclosed the fact that this patient had worked steadily for 11 years prior to the accident, that he had made no complaints and had lost no time on account of illness. He said he had never been treated for syphilis. It was impossible for me to say that the injury which this man sustained may not have activated a probably latent syphilis. When I saw him he was showing objective evidences of disease of the central nervous system. Whether these were entirely due to syphilis it was impossible to determine with certainty. This doubt is especially true of the increased reflexes on the left side and the residual left facial weakness. The fracture of the skull was on the left side it is true, but the question of brain injury by contrecoup enters here.

I am of the opinion in cases of this kind that if the complaints and the organic neurological signs occur within a reasonable time re-

lationship following the accident, say within a very few weeks, the injury may be held responsible for activating the latent syphilitic condition and localizing it in the brain. I believe that this occurs because of the increased permeability of the damaged cerebral blood vessels to the spirochete, and in this way a latent constitutional syphilis may become a cerebral lues. The experiments of Abel⁷ 17 years ago, repeated and refined by Syz^{8,9} and Dandy,¹⁰ and others recently, are the basis for this statement.

It has been held by some that in order to show the causal relationship between trauma and the onset of psychotic symptoms the relative latency period normally present between the time of infection and the precipitation of the psychosis must be shortened before the trauma may be held responsible. Otherwise, it is merely a coincidental happening. However, the latent period between the time of the primary lesion and the onset of cerebral syphilis varies so (generally from 5 to 15 or more years), that it seems to me that this criterion is of no value. A much better one is the factor of the time relationship between the date of injury and the appearance of the symptoms. If these occur reasonably soon after the injury, and further, if over and above the cerebral luetic symptoms one can demonstrate by clinical examination that focal brain lesions are present which are very likely due to trauma, then it seems to me that one must admit causal relationship.

The question of the relationship between trauma and psychoses developing following injury is often a very perplexing one. I think too many men lose sight of the fact that there is very good evidence for the belief that true traumatic insanity is very rare. All of the medical statistics, not only of the recent war, but also of the Franco-Prussian and other great wars, bear out this statement.¹¹ A brief review of the data gathered from the annual statistical study of patients in the private licensed and state hospitals in New York State for the year ending June 30, 1927, is very interesting in this connection because it gives us an opportunity to cull material from various sources and thus arrive at more reliable data. In the report mentioned, from pages 217 to 220, it would appear that with a total of 756 first admissions in all the privately licensed institutions in the state, the diagnosis of traumatic insanity was made only eight times. All of these patients were committed

and the cases classified under the New York State hospital system. On pages 180 and 181 will be found the statistics concerning out-patients examined at various clinics throughout the state conducted by the state hospitals. This gives us a valuable insight into the problem of the frequency of the traumatic psychosis in the general population outside of the state hospitals. Of 1833 cases examined (first admission to the clinic) there were 10 cases of traumatic insanity encountered. From pages 164 to 208 a careful study of the statistics shows that of the 7928 first admissions to the state hospitals the diagnosis of traumatic insanity was made in 50 cases. Referring to the statistics for the two institutions in the state which house the criminal insane, page 216, there were 185 new admissions and the diagnosis of traumatic insanity was not made in any of these cases. There was one case undiagnosed but nothing in the statistical data would lead one to suspect that the question of trauma entered into the differential diagnosis in that case. In other words, out of a total, from all the sources mentioned, of 10,702 cases, all first admissions, the diagnosis of traumatic insanity was made 68 times. Expressed in another way, traumatic insanity occurs in the State of New York in its entire in-hospital and out-patient insane population, in .0064 per cent.

The conception of traumatic insanity refers to a very definite entity. This does not mean that psychoses may not be precipitated by or the emotional reactions contributed to by injury. Nevertheless, while this is admittedly so, it is true, on the other hand, that insanity due exclusively to trauma is very rarely encountered as a result of even serious cranio-cerebral injury.

The following is submitted as an example of typical traumatic insanity unquestionably caused purely by injury:

The case is that of M. F., male, 64 years of age, a laborer, of Irish extraction, first seen on January 19, 1921.

Some time during the latter part of 1919 the patient was struck in the back of the head by a derrick chain. He was rendered unconscious, bled from the right ear and was taken to a hospital where he remained 13 weeks. He was unconscious for more than two days. He returned to work in the early part of 1920 although he was still complaining of severe headaches, restlessness at night and irritability. Several weeks after this he exhibited his first anti-social behavior. Whereas previously a mild mannered, good natured laborer who took a drink occasionally but never became drunk or offensive, he now became quarrelsome and was arrested, charged with having beaten his wife

while drunk. We were able to read the probation officer's report concerning that incident and found that just before this there had been a good deal of quarreling at home and that M. F. had been drinking heavily. This illustrates the first striking feature of these cases, namely, the deleterious effect which alcohol has on the so-called traumatic constitution. Increased susceptibility to alcohol is one of the striking features. We were never able to obtain the hospital records of this injury because when we saw M. F. in 1921 his memory was quite unreliable and he was unable to aid us.

On November 16, 1920, this unfortunate man was struck in the left frontal region by a 16-pound pile driving maul. He was unconscious for about 10 minutes and then went with the aid of his foreman and a friend to the Roosevelt Hospital where he was admitted with a diagnosis of depressed fracture of the vault of the skull. He was operated on immediately and under local anæsthesia the fragments were elevated. The hospital records say that there was no bleeding from any cranial orifice and that he made an uneventful recovery and was discharged cured December 5, 1920. When I saw him in January, 1921, he was complaining of annoying pains in the neighborhood of the injuries to the head, dizziness, a pounding headache at times, staggering gait after sudden changes of posture, irritability and fatigue. His wife said that his memory was bad and especially after he drank he became quite unpleasantly irritable.

The mental examination showed that especially when he was drinking F. was rather morose, sullen, suspicious and jealous of his wife. He was irritable with the children. The routine tests for memory showed grave defects. F. had complete amnesia for the incidents surrounding the original injury of 1919. Eventually F.'s behavior became so uncontrollable that he had to be committed to the Manhattan State Hospital where he has remained ever since. I went to visit him about two years ago at the hospital and probably entirely because he had not been able to obtain any alcohol his behavior was found to have improved but he was still unreliable, moody, depressed and occasionally quite assaultive. Shortly before I saw him for the last time he had, in one of his rages, bitten the ear of another patient. There were no delusional trends or hallucinations but he was usually quite sullen.

It is interesting to note that the conception of traumatic insanity originally advanced by Adolf Meyer⁸ in his article on the "Anatomical Facts and Clinical Varieties of Traumatic Insanity," has stood to this day without much modification. The typical case of insanity due entirely to head injury, therefore, is one in which the following sequence of events occur: First, severe cranio-cerebral injury followed by a period of unconsciousness or delirium, later recovery with grave defect symptoms, marked behavioristic changes, giddiness or vertigo, sullenness, irritability, increased susceptibility to alcohol and a psychosis which becomes usually progressively worse. There are a few points of contact between this clear-cut con-

ception of traumatic insanity and the functional psychoses or the cyclothymic reactions (manic-depressive insanity). I believe, of course, that an injury may supply the emotional factor which may precipitate a depression or an excitement in an individual possessed of the manic-depressive constitution, but these reactions should never be confused with traumatic insanity. I believe, too, that in very exceptional cases the sharp, dramatic, emotional reactions of a hallucinating or delusional case of dementia præcox may be precipitated apparently out of a clear sky by an injury, but I agree with the best psychiatric opinion that injury alone cannot cause dementia præcox. The accident and associated incidents are simply additional material at hand for elaboration and manipulation by the patient into ideas of reference and hallucinatory experiences. I say "apparently out of a clear sky," because careful investigation will invariably show that the psychiatric skies have not really been clear. It is my own opinion that an injury should be held responsible simply for the immediate precipitation of the emotional episode and should be judged in the light of a temporary influence and not by any means as the causative factor in the vast majority of these cases.

As an example of injury associated with dementia præcox the following case may be of interest:

S. S., male, 19 years of age, was injured February 26, 1926, when he struck the left index finger while at work. On February 28, 1926, the finger became infected and very painful. It was incised and dressed on March 5, 1926. According to the statement of the relatives it was at this time that the patient first began to show "nervous symptoms." The patient said, "When I got home that night (March 5, 1926) I wanted to jump out of the window." He was apparently very much upset emotionally and the next day was taken to the Presbyterian Hospital where he remained one week, and from there was removed to the psychopathic ward at Bellevue Hospital. When I saw him on March 26, 1926, the patient said, "They accused me of killing people, yet I have no tools, knives or guns. They wanted to put me away, they tied me down because I wanted to jump out of bed to get away from my enemies, etc." S. S. said that the doctors at the Presbyterian Hospital had poisoned his body by putting medicine in his sore finger. He was very emotional, restless, impulsive and had ideas of reference and delusions of persecution.

As we studied this case we found that the setting for the dramatic outbreak of the psychosis had existed for years and had gone unrecognized by the patient's relatives. He was quiet, seclusive, irritable, sensitive, would not play with other boys, had no pals and did not indulge in the usual boy's games. He stayed at home a great deal and read books and novels. He delighted his

mother by his devotion, and washed dishes and helped her with the other housework. He did not care for the movies; never smoked. His mother told with great pride how she had never heard him utter a swear word. When he grew older he always refused to call on girls with the other boys and never would take a drink. The father glowed with evident satisfaction as he boasted of what a fine boy the patient was and how he was "timid and bashful, almost like a girl." The psychogenic factors which operate in this type of psychosis were developed quite clearly when the boy told us "that at 15 years of age while I was playing marbles in a cellar alone, a man came down, tied me up, put me in a closed wagon and took me to his apartment and there he did things to me. I have walked with him many times but he disguises himself all the time and does something different each time." These homosexual ideas referred unquestionably to some previous experience, either fantasy or real, which played a great part in determining the boy's mental reactions. We also obtained the history that he had masturbated a great deal since he was 13 years of age. In speaking of this S. S. said, "Now everything in me is dried up and my spine is so dry that it crackles and breaks and hurts."

In spite of the fact that the emotional psychotic features did not make themselves manifest to his parents until the time of the injury and the infection of the finger, in all fairness it must be realized that this patient was in effect a case of dementia praecox a long time before the injury of February 26, 1926, and that the accident was simply an incidental emotional contribution which may have aided in the precipitation of the spectacular features of the psychosis simply by supplying additional material for delusional elaborations. In fact, I think we are justified in saying that his delusional handling of the finger infection episode and the consequent emotional outbreak were directly the result of schizophrenic reactions and that practically nothing was contributed by the injury to the development of the psychotic symptoms. To have charged the insurance carrier with the responsibility of looking after this patient for the many years which this psychosis will unquestionably continue, it seems to me would be unfair. The decision which was finally made was that the finger disability should be treated apart from the psychosis and an award was made for the weeks of disability generally consumed in the recovery from such an injury plus a compromise award for an agreed on period of several months which under the circumstances was quite satisfactory to all the parties concerned.

The foregoing illustrative cases are, of course, not the sole basis for certain briefly, perhaps dogmatically, stated conclusions. The results of experience in similar cases extending over a period of many years are crystallized here and the author hopes that recording his experiences and opinions may serve as an aid to those called upon to give their views, however, unwillingly, in cases of nervous or mental diseases in which trauma has played a part.

CONCLUSIONS.

The term traumatic neurosis should be limited to those cases exhibiting primitive instinctive emotional reactions to injury. These reactions may be considered as being the response of even a normal personality to fear and pain and the other acute stressful features associated with the accident. Thus the ego is overwhelmed and emotional symptoms occur.

The term traumatic hysteria should be limited to those compromise, conversion reactions of maladjusted individuals with personality and character defects often associated with physical disorders occurring following injury. In my experience, at least where litigation is a factor, these cases invariably show the so-called stigmata of hysteria. Analysis of these cases shows that the injury is very often a minor factor in the complete picture.

In this second type of case it is my experience that readjustment is hardly ever possible until the litigation is finally settled, especially so when the gainful possibilities of the situation come clearly into the patient's consciousness.

The post concussion neuroses are considered as a traumatic encephalopathy.

A head trauma may, by increasing the permeability of the cerebral vessel to the spirochete and the luetic virus, cause a latent syphilis to become active and cerebrally localized. Time relationship, the presence of focal signs and previous history of normal health are, of course, important considerations.

Psychoses exclusively the result of cranio-cerebral injury are quite rare. The concept traumatic insanity is a definite clinical entity but is very infrequently seen even with severe head injuries. The sequence of events are first, severe cranio-cerebral injury, then unconsciousness and delirium followed by slow recovery with defect symptoms, increased susceptibility to alcohol, irritability, moodiness, personality changes, vertigo and headaches, sweating or other vasomotor phenomena, etc. Trauma, however, even when not directly to the head may precipitate a depressive or manic attack in one possessed of the manic-depressive constitution.

Trauma alone cannot cause dementia præcox. When it plays a part it acts simply as one of the contributing emotional factors and its influence on the development of the psychosis is fleeting. It may

give a temporary emotional coloring to the picture and may supply material ready to hand for delusional elaboration.

In all psychotic cases one must get data of family history, personality development and the presence of peculiar traits from all possible sources, because very frequently even well-meaning people will make definite statements that the patient was perfectly well and normal in every way until the time of the accident.

BIBLIOGRAPHY.

1. Osnato, Michael: Traumatic Neurosis. *Neurological Bulletin*, Vol. II, No. 9, p. 334, September, 1919.
2. Osnato, Michael: Traumatic Hysteria. *Neurological Bulletin*, Vol. II, No. 9, p. 341, September, 1919.
3. MacDougall, William: *Social Psychology*. John W. Luce & Co., Boston, pp. 38-55.
4. Osnato, Michael, and Vincent Giliberti: Post Concussion Neurosis-Traumatic Encephalitis. *Archives of Neurology and Psychiatry*, August, 1927, Vol. 18, pp. 181-211.
5. Meyer, Adolf: The Anatomical Facts and Clinical Varieties of Traumatic Insanity. *American Journal of Insanity*, 6/288, 374, 377 and 382, January, 1904.
6. Osnato, Michael: Trauma and Other Non-Luetic Influences in Paresis. *Journal of Nervous and Mental Diseases*, August, 1920, Vol. 52, No. 2, p. 112.
7. Abel, J. J.: *Journal Pharm. & Exper. Therap.*, 1912, III, 581.
8. Syz, Hans C.: On the Influence of Asphyxia Upon the Action of Convulsant Dyes and Upon Their Entrance into the Substance of the Central Nervous System, II. *Jour. of Pharmacology and Experimental Therapeutics*, Vol. XXX, No. 1, November, 1926.
9. Syz, Hans C.: Observations on Experimental Convulsions with Special Reference to Permeability Changes. *American Jour. of Psychiatry*, Vol. VII, No. 2, September, 1927, p. 209.
10. Dandy, Walter E.: Experimental Investigations on Convulsions: Their Bearing on Epilepsy. *Journal Amer. Med. Asso.*, Vol. 88, No. 2, January 8, 1927, p. 90.
11. Trotter, Wilfred: Shell Wound of the Head. *Brain*, 42: 353, 1919.
12. Sargent, Percy: Some Observations on Epilepsy. *Brain*, 44: 312, November, 1921.
13. Cassasa, C. B.: Multiple Traumatic Cerebral Hemorrhages. Reprinted from *Proceedings of the New York Pathological Society*, N. S. 24: 101, January-May, 1924.
14. Annual Statistical Review of Patients in the State Hospitals and Private Licensed Institutions for Mental Disease. For the Year ended June 30, 1927, p. 208 (Publisher J. B. Lyon Co., 1928).

DISCUSSION.

DR. ADOLF MEYER.—The time is so very late that I should like to make only two remarks. In the first place, the whole chapter of traumatic reactions of the neurosis and of the psychosis type is exceedingly complex and really one in which it is practically impossible to make any concise groups. At any rate, evidently, a very large factor is the psychic, the personality reactions, and equally important, and naturally from the scientific point of view and ultimate judgment point of view, the structural problem is an exceedingly difficult one to gage. In the specimen that has been shown us, there comes to the front something that has been particularly well discussed by Dr. Bagley, the brain surgeon, in Baltimore, who has worked out his traumatic material in our laboratory. I am convinced the case shown us is one in which the traumatization of the corpus callosum, that is to say, an injury with impaction, by the falx, evidently played a very important part in contusing the corpus callosum and producing the numerous hemorrhages which besides the other lesions require quite a good deal of attention. Those cases behave in a very interesting way from the point of view of either the collapsing of the brain or the swelling of the brain, a point which is very well discussed in that paper of Dr. Bagley's. I think it is very opportune that such a review as Dr. Osnato has given us may start again a rejuvenation of the discussion of this very important topic.

DR. J. KASANIN (Boston, Mass.).—Dr. Osnato's concept of traumatic encephalitis seems to be a very interesting concept and merits a great deal of attention. I had the opportunity of going over about 15 or 20 cases of children who have had early cerebral traumas, either a serious concussion of the brain or a fracture of the skull. It was interesting to know that following these injuries, the children have shown the same sort of a clinical picture as the cases who have had encephalitis, that is, it was a very marked personality change in the sense of marked emotional stability, inability to follow any goal in life, marked irritability, together with other symptoms which are peculiar to the traumatic cases, that is, inability to stand in shut-in, closed-in spaces, and to withstand hot weather. The only difference in these cases as compared with the cases of epidemic encephalitis was the question of prognosis. Following three or four years after the injury, there was a marked tendency toward improvement in the general condition of the patient, with a fairly satisfactory adjustment in society, while the cases of epidemic encephalitis did not seem to show an equally good prognosis.

THE METABOLIC RATE IN EMOTIONAL MOODS INDUCED BY SUGGESTION IN HYPNOSIS.*

By J. C. WHITEHORN, M. D., HELGE LUNDHOLM, PH. D., AND
G. E. GARDNER, A. M.

*(From the Chemical and Psychological Laboratories of McLean Hospital,
Waverley, Mass.)*

The purpose of this communication is to report some observations concerning the relation between emotion and metabolism.

Those who have had practical experience in the determination of basal metabolic rates are familiar with the difficulty in getting reliable figures on excitable persons. It is, indeed, not uncommon to find, even with quite normal subjects, that the metabolic rate is higher on the first test, when the subject is somewhat apprehensive, than on later occasions. There is probably little doubt, therefore, among those familiar with such tests, that a person's metabolic rate is related in some way to his or her emotional state. Yet the voluminous literature on basal metabolism includes very few studies bearing directly upon this relationship, probably because few laboratory workers have cared to write for publication on a matter so vague and unmeasurable as emotional states.

Patients in a mental hospital are not, as one might suppose, good subjects for such a study, because the very factor which one wishes to investigate usually precludes a basal metabolic rate determination, *i. e.*, the emotional state prevents such subjects from keeping quiet for a period long enough to make proper tests. One of us has, however, occasionally made satisfactory observations on a patient in a state of considerable anxiety and apprehension, but able to keep physically quiet, whose metabolic rate was about 20 per cent higher than when emotionally calm. Even in such instances, however, the interpretation is not quite clear, for the emotional state and the metabolic rate may have undergone a simultaneous alteration by a third factor.

* Read at the eighty-fifth annual meeting of The American Psychiatric Association, Atlanta, Ga., May 14, 15, 16, 17, 1929.

We have, therefore, sought less ambiguous results in the study of emotional states induced by hypnotic suggestion following the lead of Grafe and his collaborators at Heidelberg.¹ As it is necessary in such tests to keep the subject physically quiet, we decided to avoid suggesting emotions with definite ideational content, for fear the subject might struggle or show other muscular response. Instead, we attempted to induce *moods*, that is, emotional states without definite objects. The hypnotic suggestions also included an injunction to keep physically quiet. These precautions sufficed in all but one experiment, which is excluded from the present report. In this instance the subject was specially requested to breathe very regularly, with the result that his mood (anxiety) became focussed on the apparatus and he struggled to free himself from it. In the other experiments, which were accounted successful in this respect, the only muscular movements were changes in facial expression and an occasional motion of the lips or fingers. The hypnotic suggestion of a *mood* removes also some of the ambiguity regarding the kind of emotional experience induced in experiments like those of Grafe and Mayer.¹ For example, if the subject is led to believe, by hypnotic suggestion, that he is in a fight with cannibals, he may experience either fear or anger or both. Our mood suggestion should, therefore, enable us to control with somewhat greater certainty, the type of emotional experience induced. This point has also been checked by the subject's retrospection.

PROCEDURE.

The subject of these experiments was a man of 24 years, physically and mentally normal, height 175 cm., weight 63 kg. \pm 3 kg., a candidate for the Ph. D. degree in psychology, and an excellent subject in hypnosis. A series of basal metabolic rate determinations was made on him for a few weeks before the hypnosis experiments began, in order to get him accustomed to the tests and to establish a base line. Unfortunately his rate varies somewhat from week to week. On this account every observation in a mood period was compared to a control test immediately before or after, in hypnotic sleep. His average basal metabolic rate was 225 cc. oxygen per

¹ Grafe, E., and Mayer, L., *Zeitschrift f. d. ges. Neurol. u. Psychiat.*, lxxxvi (1923), 247, and Grafe, E., and Traumann, *Zeitschrift f. d. ges. Neurol. u. Psychiat.*, lxii (1920), 237.

minute (36.5 Cal. per sq. m. per hour, which is 8 per cent below the Aub-DuBois standard). The procedure for an experimental period was as follows:

The subject, who had taken no food for 14 hours, lay quietly at rest for half an hour, about 7.30 a. m. to 8 a. m. He was then brought into hypnotic sleep (by L.) and the following suggestion was given to him:

I am going to wake you up. When you have been awake a few seconds I raise my finger in front of your eyes. As I raise my finger you will immediately go into a very deep and quiet sleep. This sleep will be dreamless, and just as deep as the deepest normal sleep. It will last until I, by counting to three, wake you up. The moment you wake up there will come over you a peculiar feeling of intense anxiety. It will seem to you as if a danger is threatening you. The nature of this danger you will not know, but yet you will suffer distinct emotional tension and apprehension. In spite of this tense state, you will control your overt motion for the sake of the experiment. The compulsion to remain apparently quiet will intensify your anxiety. After a certain length of time I will suddenly relieve you of this emotional condition by raising my finger in front of your eyes. As I do this you will immediately be relieved and feel very well. From now on, you will not remember anything I have told you during your sleep, yet you will act in accordance with it. I count to three and on three you wake up.

The subject was then awakened by the operator's signal (counting to three), and connected (by W.) with a metabolism apparatus of the Roth type by means of mouthpiece and nose clip. After the few seconds required for this adjustment, the sleep signal was given (raising the finger) and the subject immediately went back into sleep. The rate of oxygen consumption was graphically recorded during this control period of hypnotic sleep and during the mood period which followed, according to the suggestion. (The technical checks against leaks and incomplete carbon dioxide absorption were performed in the usual way by weight on the spirometer and by barium hydroxide solution.) During the final sleep period (see suggestion) the subject was instructed (by L.) to remember the details of his experience. After being awakened and disconnected from the apparatus, the subject wrote out a retrospective account of his experience. One such account of the anxiety mood reads:

My feeling was that of fear, though all attempts to localize the source of threatening danger were futile. At intervals there seemed to pass through my mind pictures of danger-filled situations and for a time it seemed as if a

large rock was descending upon me. All attempts at movement seemed to be cut short at their inception by some counter impulse. These "pictures" were merely momentary flashes.

In the following paragraphs are given some retrospective accounts of other moods, together with the salient points of the suggestions which evoked these moods:

IIa. *Suggestion for Depression*.— . . . a peculiar feeling of profound depression. Everything will seem dark and dull to you and you will feel as if none of your ambitions or aspirations will come true. There will be no apprehension connected with this mood, it will be just a feeling of calm and hopeless resignation and depression. . . .

IIb. *Retrospection on Depression*.—To-day's session was marked by an intense feeling of sadness or blueness with inability to localize the cause of the feeling. There was no kaleidoscope of pictures corresponding to the general mood. It was rather a feeling of futility. It was an effort to breathe and at times it seemed I had to force myself to do so. There seemed to be no desire to escape, just a continual thought of "I wonder why I am so sad."

IIIa. *Suggestion for Elation*.— . . . a peculiar feeling of profound happiness and elation. It will seem to you as if you are all-powerful and as if all your desires are to be gratified. . . . You will remain quiet on the bed, just enjoying your elated and happy mood. . . .

IIIb. *Retrospection on Elation*.—There seemed to be an urge to activity of some sort. I wanted to be in action but these impulses were continually cut short, resulting in mere movements of the fingers and toes. There was a peculiar feeling of joy and carefree-ness which was not, however, determined by any particular idea or group of ideas. I sensed, in addition, a "pressure" in the chest—a condition best described as a "tightening under tension."

IVa. *Suggestion for Irritability*.— . . . a peculiar feeling of extreme irritability. It will seem to you as if everything and everybody is thwarting you and is set on evoking your anger and irritation. You will feel very touchy about this matter. Yet there will be no specific object of your irritation. . . .

IVb. *Retrospection on Irritability*.—Thoughts were of petty annoyances of various kinds, though the period to-day was marked by a generalized dislike for all people in general and for the experimenters in particular. My strongest impulse was to get up and leave the metabolism room, and in so doing to show my contempt for the proceedings. Upon trial, however, I found that this was impossible. I remember particularly that I saw no reason why my pulse should be taken. In short it was anything but a desire to cooperate with people.

Similar experiments were also conducted in reverse order, by re-arranging somewhat the wording of the suggestion, to get the metabolic rate of a mood period *before* that of a control period.

TABLE I.

Protocol No.	Post-hypnotic mood.	Metabolic rate cc. O ₂ /min.			Pulse rate per min.			Respiration in mood period.
		Mood period.	Control period.	Increase.	Mood period.	Control period.	Increase.	
6	I. Anxiety before sleep.....	240	214	26	76	67	9	Irregular.
7	Anxiety after sleep.....	289	251	38	80	70	10	Irregular.
15	Anxiety before sleep.....	283	221	62	80	68	12	Very irregular.
16	Anxiety after sleep.....	280	210	70	80	72	8	Slow and irregular.
18	Depression after sleep.....	218	214	4	64	64	0	Slow and irregular.
19	Depression before sleep.....	232	214	18	60	60	0	Very slow and irregular.
20	Depression after sleep.....	227	243	-16	76	72	4	Slow and very irregular.
11	Elation before sleep.....	204	203	1	74	67	7	Rapid and shallow.
12	Elation after sleep.....	248	267	-19	72	64	8	Irregular, mostly rapid and shallow.
17	Elation after sleep.....	247	220	27	76	72	4	Irregular and rapid.
13	Irritability before sleep.....	228	237	-9	74	64	10	Irregular.
14	Irritability after sleep.....	263	242	21	84	68	16	Irregular.
1, 2, 3, 4, 5, 10	Basal metabolic rate without hypnosis, average		225			67		
9, 10	Abnormal breathing experiments, average		223			74		

Average increase, Anxiety, 49 cc., 22%.
 Average increase, Depression, 2 cc., 0.9%.
 Average increase, Elation, 3 cc., 1.3%.
 Average increase, Irritability, 6 cc., 2.5%.

DISCUSSION OF RESULTS.

Our observations are recorded in Table I. We should have liked to include the blood pressure and chemical analysis of the blood, but did not do so, for fear of disturbing the hypnotic condition.

Both pulse and respiration were altered during mood periods. The pulse rate increased slightly as a rule. The respirations, which were recorded graphically by the Roth apparatus, showed very marked changes in rate and depth. In fact the irregularity of breathing frequently made it impossible to estimate the rate of oxygen consumption closer than 5 or 10 per cent. On this account, no definite significance can be attached to apparent changes in rate under 10 per cent. We were unsuccessful in our attempt to cut down this error by hypnotic suggestion to breathe very regularly. We are able, however, to satisfy ourselves by appropriate control experiments not described in this report, that the types of respiration encountered in this study do not, of themselves, alter the rate of oxygen consumption.

With these considerations in mind, it is clear that the apparent slight increases in metabolic rate observed in the moods of elation (1.3 per cent), irritability (2.5 per cent) and depression (0.9 per cent) are too small to mean anything. On the contrary, there is a marked increase (22 per cent) during the mood of anxiety.

CONCLUSIONS.

The mood of anxiety or apprehension, hypnotically induced, can increase the metabolic rate.

Moods of depression, of elation and of irritability, hypnotically induced, have not, in our experiments, produced any certain increase in metabolic rate.

WIDESPREAD PRESSURE ATROPHY OF THE BRAIN,
AND ITS PROBABLE RELATION TO THE FUNC-
TION OF THE PACCHIONIAN BODIES, AND THE
CEREBROSPINAL FLUID CIRCULATION.* †

By TEMPLE FAY, M. D.,

*Professor of Neurosurgery, Temple University School of Medicine;
Neurosurgeon to the Episcopal, Philadelphia General
and Jewish Hospitals.*

AND

N. W. WINKELMAN, M. D.,

*Professor of Neurology, Temple University School of Medicine;
Neuropathologist of the Philadelphia General Hospital.
Philadelphia.*

The roentgenographic studies of the brain following encephalography¹ have presented a variety of findings requiring interpretation. The close analysis of the clinical history and neurological examination has not sufficed to explain some of the conditions noted.

In a series of approximately 200 cases, increased collections of air have been noted in various positions within the subarachnoid

*This paper is the second of a series on the pathological and clinical studies in Epilepsy. (1) Some factors in the "Mechanical theory of Epilepsy," with especial reference to the influence of fluid, and its control in the treatment of certain cases, Fay, Temple, *Amer. J. of Psych.* 8; 783-833 (Mar.), 1929. (3) The Pacchionian System; Histologic and pathologic changes with particular reference to the idiopathic and symptomatic convulsive states, Winkelman, N. W., and Fay, Temple, *Arch. Neurol. and Psychiat.* 23: 44-64 (Jan.), 1930. (4) Encephalography; Roentgenological and clinical considerations for its use. *Amer. J. Roent. Rad. Therap.*, 21; 421-447, (May), 1929. (5) Generalized pressure Atrophy of the brain secondary to traumatic and pathologic involvement of the Pacchionian bodies, Fay, Temple, *J. A. M. A.*, 94; 245-250 (Jan. 25th), 1930. (6) The results obtained by dehydrating epileptics, Fay, Temple, *Arch. Neurol. and Psychiat.* (in press) 1930.

† Read at the eighty-fifth annual meeting of The American Psychiatric Association, Section on Convulsive Disorders, Atlanta, Ga., May 14, 1929. From the Daniel J. McCarthy and Memorial Foundations, Temple University, the University of Pennsylvania and the Laboratory of Neuropathology of the Philadelphia General Hospital, Philadelphia, Pa.

spaces, and over the cerebral hemispheres, as well as in the various cisternæ. For want of a better term these areas which represent increased fluid collections have been designated as being evidence of cerebral "atrophy." The findings have been most numerous in the post-traumatic chronic epileptic, and senile degenerative types, but large areas of "atrophy" have also been noted in infants as early as the third month, and the need for determination as to the character and cause of this loss of cerebral volume has prompted the following consideration on this subject:

Cerebral atrophy has long been recognized as a sequelæ of thrombosis producing areas of softening. The shrinkage and contraction of the brain substance following such vascular lesions have permitted the accumulation of cerebrospinal fluid to replace the spaces left by such an atrophy. The considerations governing this "space compensation" mechanism are fully taken up by Dandy.* Following trauma, associated with either direct interference of vascular supply or actual contusion of the brain substance, as well as multiple punctate hemorrhages in the subcortical areas, there may be a replacement of brain substance by glial and scar tissue, and later, actual contraction representing another type of cerebral "atrophy." Recently, Hassin¹² has demonstrated a type of "pressure atrophy" which occurs in the neighborhood of solid cerebral tumors. The atrophy produced by internal hydrocephalus is well known to all, and that there may be a similar atrophy produced by external hydrocephalus has also been recognized. The mechanism and characteristics of this latter type concern this presentation.

In the analysis of approximately 200 cases of encephalography, there were found a large number of unexplained "atrophies," without history or findings of a vascular lesion; frequently without sufficient neurological evidence to explain the gross presence of this atrophy where trauma, syphilis, or meningeal infections have not played a rôle. This group of cases strongly simulate *in vivo*, by stereoscopic comparison, those brains in the pathological collection classified as, "aplasia," "agenesis," "idiopathic atrophy," infantile and undeveloped hemispheric characteristics. The small, nodular contracted convolutions sometimes focal to a definite area, representing an isolated lesion, or widespread involvement in the frontoparietal area, appear to be demonstrable on the stereoscopic roentgenographic films, and it seems fair to assume that the charac-

teristics, where noted, would probably closely correspond to those specimens now in the laboratory, which have had no adequate explanation of such widespread pathology in the past.

Neuropathological studies were undertaken to correlate the findings in the pathological specimens representing the various types of atrophy. It has not been possible to obtain more than two specimens in which the patients were subjected to encephalogram, and their brains examined following the findings recorded on the roentgenographic films. Fortunately, encephalography has not had, in careful hands, any lethal effect upon patients, excepting those with brain tumors. Hence the comparison has required correlation with miscellaneous specimens simulating the gross changes noted by encephalogram, a more direct comparison at this time being impossible. It is at once evident that the areas of atrophy do not conform to any arterial cerebrovascular circulating plan. In many of the cases the atrophy fuses over two or even three arterial supplies without having given evidence of vascular dysfunction in the patient's history or clinical findings. It is for this reason that the idea of thrombosis as an underlying factor has been abandoned, excepting in those cases in which a definite vascular insult was clearly defined by the history or clinical findings. Furthermore, the appearance of this widespread atrophy in infants precluded the possibility of vascular insults at an age when thrombotic lesions are not frequent. Trauma has been associated with a surprisingly large number of these cases, if we include in trauma the possibility of birth injuries and secondary meningeal inflammations occurring at any period of life.

A rather striking fact is that in cases of generalized "atrophy" the encephalographic findings closely simulate those of the gross specimens, in that the frontal and parietal lobes have suffered most in this process. Dandy⁷ has called attention to the fact that fluid collections have not been met with over the temporal lobe or the under surface of the frontal lobes, and only occasionally over the occipital lobes of the brain, in his experience with epileptics and others, showing increased fluid accumulations. This fact is even more striking when one attempts to explain, on a vascular basis, the escape of the temporal lobe, or the occipitoparietal area, and it becomes even more difficult when "atrophy" of the cerebellum is associated with generalized frontal atrophy. A careful study,

however, of Fig. 1, representing the normal cerebrospinal fluid circulating field, strongly suggests that the most pronounced types of atrophy are seen within this general field of cerebrospinal fluid circulation, and as cerebrospinal fluid accumulations have been one of the outstanding facts associated with this type of atrophy, one may well ask the question as to whether some dysfunction of the cerebrospinal fluid circulating mechanism may not be directly or indirectly responsible for the changes noted by encephalography, and seen in the gross specimens under consideration. The fact that cerebrospinal fluid may not find a normal means of escape sufficient to meet the rapidity of its formation has led to the consideration as to whether these increased fluid collections may not, by their presence and pressure, produce, over long periods of time, generalized atrophy confined to the usual cerebrospinal fluid circulating fields.

It is striking that the brains of mental defectives have been frequently characterized by this marked loss of cerebral volume in the frontoparietal regions. Encephalographic studies on epileptics have given the distinct impression that, in the early stages of the disease, the areas of increased fluid collection are not marked, and are usually characterized by small areas situated at the vertex or in the frontoparietal surface. However, those cases which have progressed throughout a period of several years, and in which frequency of attacks is associated with definite mental deterioration, there appears a striking contrast in that large areas of generalized shrinkage of the brain have been noted.

The progressive mental deterioration has been accounted for on the ground of actual cerebral volume loss, and the areas most affected are those of the frontal and parietal regions.

The finding of increased amounts of cerebrospinal fluid in the epileptic has been a common experience by many observers, even from the time of Hippocrates. The part that this fluid accumulation may play in producing a progressive type of atrophy, similar to that seen in the mentally defective without convulsions, becomes of prime importance.

Weed^{19, 21, 22, 23, 24} has brought forth the most convincing evidence so far offered, that cerebrospinal fluid is eliminated for the most part by the subarachnoid villi or the pacchionian bodies. The fluid itself responds to the laws of a dialysate as pointed out by Fremont-

Smith¹¹ and probably dialyzes from the subarachnoid space into the large venous channels at the vertex, as well as elsewhere, wherever subarachnoid villi or pacchionian granules may occur. The elaboration of cerebrospinal fluid is thought for the most part to be a transudate of this fluid from the blood plasma through the choroid plexus in the lateral and fourth ventricle though undoubtedly fluid also arises in the perivascular spaces themselves. The work of Kubie¹² and Howe¹³ adds strength to this possibility. As a large portion of cerebrospinal fluid originates within the ventricles, it finds its escape through the foramina of Luschka and Magendie into the subarachnoid space of the posterior fossa. It then passes anteriorly through the cisterna pontis, and the narrow constriction caused by the tentorium, into the cisterna chiasmatis, and thence it is distributed over the cerebral hemispheres, the usual pathways being anterior in the interhemispheric aspect over the corpus callosum, or along the sylvian fissure to find its way by devious channels over the frontal and parietal lobes to the region of the pacchionian bodies, situated along the longitudinal sinus. (Fig. 2.)

We may therefore expect that fluid having reached its point of elimination, and finding a deficient mechanism as described by us,² might accumulate first at the vertex and then by back-pressure, along the devious channels existing over the frontal and parietal areas to the cisterna chiasmatis. Indeed this is the impression that many of the encephalographic findings denote. Although the analysis is crude, still the body offers no other similar type of change than that seen in Fig. 3, in which obstruction along the urinary system discloses the dilatation due to obstruction affecting all of the structures within the system, with marked involvement of the areas closest to the point of occlusion.

That pressure atrophy of this character may occur is conceivable in the same light that a snug fitting cast applied to an extremity produces in two to three weeks definite signs of loss of tissue volume which we usually term atrophy. In the frontoparietal areas, there is in reality a close fitting covering of fluid which may act, with pressure, similar to the application of a cast, especially if gradual, long-standing accumulations of this fluid have taken up all of the available compensatory space within the closed confines of the skull. Coughing, straining, epileptic seizures, hypertension

and overindulgence in fluids, have all been associated with increase of intracranial pressure.

It has been the opportunity of one of us (Fay) to view, on three occasions, a convulsive seizure, with the brain exposed. The pressure during a convulsive attack is so great that the brain protrudes from the opening in the dura, and may even rupture with the volume of its swelling. This observation has been noted by Foster Kennedy⁹ as well as by Foerster.^{9, 10} We are all familiar with the Queckenstedt test and the changes in pressure which occur during occlusion of the jugular, and in coughing and straining responses. It is therefore possible to find means by which intracranial pressure might be accentuated during certain periods. There is undoubted evidence that following an acute, traumatic lesion, or with alcoholic wet-brain and meningitis, increased intracranial pressure is a frequent finding. The effects of these pressure mechanisms on the subarachnoid systems are similar to those produced in other closed systems, following the law of hydrodynamics. These factors are taken up in detail in a paper by Pancoast and Fay¹¹ who concluded that, with a defective system of elimination, increased subarachnoid accumulations of fluid may occur, giving rise to focal as well as generalized areas of atrophy, depending upon the extent and character of the pathology as well as its location.

The effect of long standing, intermittent phases of pressure upon the cortical, capillary circulation would appear to be sufficient, so that normal development of the cortical areas would be impaired, and even a process of atrophy favored when such pressure mechanisms existed.

Following subarachnoid hemorrhage, either spontaneous, post-traumatic or after birth, Weed¹² and Bagley² have shown a specific reaction of the arachnoid to red blood cells. A tendency to delay, impede and obstruct cerebrospinal fluid circulation during the period of ten days or more of absorption may give rise to an increase in intracranial pressure, and it is during this phase that great damage may be done, so that in several cases in our series, it seems that the acute pressure phenomena occurred only for a short time, and was similar to that experienced in meningitis, leaving behind the possibility of loss of brain tissue and subsequent so-called cerebral "atrophy."

With these possibilities in view to explain a type of atrophy so widespread, studies have been conducted to determine if the characteristics of this atrophy could be associated with vascular disease, *per se*, or whether these changes might have been caused by the presence of abnormal collections of fluid, under pressure, unable to find a proper means of escape, and thus account for the characteristic changes noted in many of the mentally defective and chronic degenerative brains found in almost every neuropathological collection.

It is important to consider the factors which may underly the delay in cerebrospinal fluid circulation, or the abnormal accumulations of this fluid, due to some deficiency in the mechanism of elimination.

We are reporting before the American Neurological Association (1929) the results of study of the normal anatomy of the arachnoid villi and the pacchionian granulations. (Fig. 13 *a* and *b*.) We have also gone into the different types of pathological changes that may occur in them, and have come to the conclusion that because of the fact that the pacchionian granules are in direct anatomic connection with the subarachnoid space, they are subject to the same conditions present within the fluid spaces of the brain and cord. We have shown that in paresis, in which fibrosis of the soft membranes is a common condition, there is likewise fibrosis of the pacchionian granulations. It has also been found that in acute alcoholism the edema of the brain and meninges is accompanied by edema of the pacchionian bodies, and likewise in meningitis where the subarachnoid space is filled with inflammatory elements, there may also be found the same exudative cells in the pacchionian granules.

With this brief summary, it is only necessary to state that we have divided the changes found into five distinct groups:

- (1) Aplasia.
- (2) Hypoplasia.
- (3) Hyperplasia.
- (4) Fibrosis.
- (5) Infiltrative.

(1) In the *aplastic group* (Fig. 14 *a* and *b*) we placed those in which there was almost complete absence of the adult pacchionian

granulations, but with the presence of arachnoid villi. (Fig 15.) As is known, this is the usual condition in childhood, but normally at puberty development into the adult type takes place. This lack of development has been observed by others, and commented on by Cushing in some of his cases of "idiopathic" hydrocephalus. In our own work a complete absence of the pacchionian system has also been found in the so-called idiopathic epilepsies, in which also an infantile appearance to the whole brain is present.

(2) In the second group, the *hypoplasias*, there was present the adult form of pacchionian tissue, but it was so shrunken that they appeared only as compact groups of mesothelial cells (Fig. 16) without differentiation into the normal structures. It may be stated in passing that a few of our epileptic cases have had this type of development.

(3) In the *hyperplastic group* there was dilatation of the pacchionian system which had been found in conditions where increased functional activity was present, as in acute alcoholism and uremia. In this there may be enormous spreading out of the individual granules with marked increase in the central core of spongy tissue and spreading out of the individual fibrils. (Fig. 17.)

(4) In the *fibrotic type* (Fig. 18) we placed that group in which the same sort of fibrosis occurred that we find in the pia-arachnoid in such conditions as paresis, chronic alcoholism and severe arteriosclerosis of the brain. With this form, characteristic degenerative manifestations may be present such as the occurrence of calcified bodies, made mention of particularly by Cushing and Weed. It is in this form especially that there is present a cortical atrophy of more or less severe degree. It is rather interesting that we find this fibrosis of the soft meninges and of the pacchionian system in association with accumulation of lakes of subarachnoid fluid with an underlying cortical atrophy.

(5) In our final division, the *infiltrative*, we found the same type of abnormal elements that were present in the subarachnoid space. We have reference here to the invasion by inflammatory elements (Fig. 19) such as polynuclear and lymphocytic elements, by blood cells and blood pigment and rarely by cancer cells.

CORTICAL ATROPHY.

Cortical atrophy (Fig. 20) is not an unusual finding in certain conditions. This is particularly true in paresis, chronic alcoholism,

epilepsy and in senile and arteriosclerotic conditions. This finding of atrophy in the above conditions has been taken as a matter of course, and it has been felt that it was the result of degeneration in the brain, with shrinkage of tissue substance. The fact that it occurs in certain regions of the brain has been commented on by many, but no studies have been made, as far as we know, to determine why it should occur in these areas. In conditions in which sclerosis of the vessel occurs the assumption has been made that the atrophy was the result of nutritional disturbances. Why the atrophic process should be confined, as it usually is to certain areas, has had no explanation. Its occurrence in conditions not associated with arteriosclerosis, as in comparatively young epileptics, is therefore without explanation. McCarthy¹ in his work on cases of pulmonary tuberculosis found "some degree of atrophy in nearly all the brains examined." His findings were that it occurred mainly in the motor area and in the anterior portion of the frontal lobe. His microscopic studies showed "rarefaction of the tissue."

In our own work we too have found the cortical atrophy more common in certain areas of the brain. Of these the frontal area is by far the most frequently involved (Fig. 5), next comes the anterior and upper part of the parietal region. We have not had that rare condition in which the atrophy has been most intense in the left temporal lobe, or what is known as Pick's disease (Altman¹). In cortical atrophy there is usually noted a depression of the convolutions, widening of the sulci, collection of fluid in the subarachnoid space above it, and a fibrosis and milkeness of the pia. (Fig. 21.) This is the condition to which the name of external hydrocephalus has been given, and is mentioned by Dandy, Foerster, Foster Kennedy and others, who have viewed the brain at operation. This is in contrast to areas, the result of softening of tissue from vessel occlusion which is more likely to be circumscribed, occurring in any part of the cortex, obliterating the anatomic landmarks in the region involved, and showing a crater-like depression. In the fairly recent state a yellowish or brownish discoloration may be present. This is the so-called *jaune plaque* of the French. (Fig. 22.)

An effort to investigate some of the problems connected with cortical atrophy has led us to a study of brains of patients who have shown atrophy conditions of various sorts. Our method of study has been as follows: We have studied 15 cases in which

arteriosclerosis was absent or very mild, and have made comparative sections from the atrophic and non-atrophic areas of each brain, and have contrasted them section for section *in each brain*. We have used in this study not only the usual ganglion cell and fiber stains, but also the newer Spanish staining methods. Our results have been highly interesting. As a comparison we have studied those portions of the cortex in cases in which vascular degenerative lesions have occurred, and we have also had at hand cases in which slight cortical depressions and atrophy have been made by solid masses such as fibroblastic tumors, as has been done recently by Hassin¹² with very interesting findings; also by gross epidural and subdural collections of blood; and finally as a result of subarachnoid accumulation of inflammatory elements as in a case of undrained meningitis.

In sections taken from the atrophic areas one notes on gross inspection of even the cell preparations that there is a marked widening of the sulci between the various convolutions, and the individual convolutions themselves are shortened and narrowed. The gray matter is narrower in this region when compared to the non-atrophic areas in the same brain, but it is not only the gray matter that is narrow but the white matter partakes of the atrophy. The ventricle is usually widened but there may be found no change in the ependymal lining.

Under the microscope the following features are worthy of consideration: The pia is usually definitely thickened and fibrotic and in contrast to that over softened areas, the result of vessel occlusion, contains few or no pigment-laden phagocytic cells, and no inflammatory elements. Its vessels show no changes.

On examination of the cell pictures of the cortex (Fig. 23) under low magnification in the severely atrophic areas, one is struck by the narrowness of the cortex in contrast to the usual picture and definitely in contrast to the more normal areas of the brain, although the architectural arrangement into layers is within normal limits. In a careful examination as to the reason for this atrophy it is noted that while there is a fairly uniform narrowness of the individual layers, especially the upper three, it is particularly in the third cortical layer that the process reaches its maximum. It must be stressed, however, that there is no gross absence of tissue such as is met with in complete vascular occlusion, with glial cell accumu-

lation, etc. On the contrary, it is very similar to changes met with in partial shutting-down of the circulation. The lowermost three layers of the cortex show a pushing together of all the elements, so that they may actually appear to be denser and richer in cells. The outer cortical layer, as a rule, is much richer in cells than is usual, and they are usually found to be mainly astrocytic, but macroglia also occur. The blood vessels, as a rule, show no changes except a relative increase.

The minute changes are of interest. The ganglion cells, as explained above, were lessened in number, but those that remain show various types of degeneration, mainly of the atrophic and ischemic varieties, and Bielschowsky preparations show a slight clumping of the intracellular fibrillæ.

Special stains made of these atrophic areas show first of all a marginal gliosis, varying in amount with the degree of pressure. (Fig. 24.) The Cajal preparations show a marked gliosis of the fibroblastic type. While fairly uniform throughout, the process was probably more marked in the outer half. The white matter beneath shows just as marked a gliosis, if not more so than the gray. In cases in which the gross atrophy was even more marked, there were degenerative changes in the macroglia, as shown by breaking up of the cell processes (clasmotodendrosis). Hortega preparations showed nothing remarkable.

Fat stains, and especially those sections counter-stained with the Morgan stain, gave us a good inkling how little fat was present in the tissues, and even in the perivascular spaces. The amount in the ganglion cells was usually commensurate with the age of the patient.

Bielschowsky preparations gave no indications of plaque formation. The intracellular fibrillar showed at times slight clumping, but no Alzheimer changes were noted.

A condition present in some of our cases, the significance of which might be better interpreted in the light of Weed's²³ and Kubie's¹⁸ work, is dilatation of the perivascular and pericellular spaces. In some cases this was a uniform finding through the entire brain, but in many cases was a focal finding in areas of marked atrophy.

It must be stressed at this point, that no focal degenerative areas occurred in these cases. The pathology fitted in, however, with what the Germans have called *verödung*.

Globus,¹¹ in his study of the glia in arteriosclerosis in which atrophy of convolutions is the usual finding, noted variations in quantity of astrocytic cells, but made no comparisons to determine the relation to the more or less atrophic regions. He found, as we did, that it at times reached the degree of glial hyperplasia found in general paralysis. In the subcortex, he also found great numbers of fibroblastic astrocytes, also variable in various regions. In severely atrophic focal areas, such as are met with in particular in Pick's disease, the changes are very similar to what we have found in the frontal lobes in our cases.

As a contrast to the atrophic areas, sections were taken from the same brains in the non-atrophic areas and stained by the same methods. While in many cases a slight increase of fibroblastic astrocytes was present, in no case did they approach the degree of proliferation seen in the atrophic areas. In many cases in which arteriosclerosis was mild, there was practically no glial increase at all. For the most part no architectural changes were to be made out, and the ganglion cells were within the limits of normal. The stain by which a differentiation could be made at one glance was by the Cajal gold-chloride method, and in many cases the difference was as marked as is represented in Fig. 25, taken from the non-atrophic area, and Figs. 26, 27, 28 and 29, taken from the atrophic region, in both white and gray matter.

In areas slightly depressed as a result of gross pressure by a small endothelial tumor or over larger areas of the cortex by an overlying subdural hematoma, one notes that the normal anatomic markings of the brain surface can be made out, but the tumor or hemorrhage has made a sort of nest for itself. On microscopic examination one finds a narrowing of the cortex as a result of compression of each of its constituent layers. There is a decrease in the number of ganglion cells in proportion to the degree of compression. The remaining ganglion cells, again, show involvement in proportion to the amount of compression, although ischemic cell disease is present to a marked degree. The Cajal stains show an increase in the number of fibroblastic macroglia which may approach that seen in paresis. The vessels appear increased in number owing

to the compression of tissue. No actual areas of softening are found and here again the picture found might be called *verödung*.

In many areas a marginal gliosis is a feature. Fat stains in cases of mild compression have shown but little, but in severely affected regions the amount of lipoid present is out of proportion to the age of the individual. Bielschowsky preparations have shown nothing in the mild cases to clumping of intracellular neurofibrilla, but no plaques.

In the cortical area of necrosis, the result of gross vessel occlusion of the *jaune plaque* (Fig. 22) of the French is seen grossly to be a brownish-yellowish crater-like depression in the cortex and in the case studied for this investigation, it measured 2 cm. in diameter and was 3-4 mm. deep. The edges were ragged. On cutting through the area one notes that liquefaction of tissue and cyst formation had occurred not only in the gray matter, but extending down into the white matter with a thin shell of pia forming a covering for this cyst-like area; the contents consisting of a brownish fluid material.

On *microscopic examination* there is noted a crater-like area in the base of which is still present innumerable fat-laden gitter cells. This is very clearly brought out in the fat preparation, counterstained with Morgan's method, and shows it to be fairly sharply margined, although gradual extension into the white matter is visible. (Fig. 29.) There is the mild collaring of vessels in the neighboring well preserved tissue, with extensions in finger-like projection in various directions. One cannot make out any remains of the cortical tissue in this area, and even the subcortex is affected, as shown not only by complete disintegration of the subcortex adjacent to the gray matter, but also a mild status spongiosus of the underlying tissue. The meninges form the covering for the cystic area, and are markedly thickened and fibrotic, and contain numerous fat and pigment-containing phagocytic cells.

In cortical regions that are atrophied, the result of numerous minute areas of partial or complete softening (*e. g.* the result of thrombi from an endocarditis), there are small foci which produce but slight gross atrophy of the brain. Microscopically the picture is that of minute focal areas of necroses of various stages, with incomplete and complete softening.

In a recent case of acute epidemic meningitis, unrecognized and untreated for several days, while the patient was in jail, there were marked changes in the cerebral cortex, that were very similar to those seen as a result of pressure. They will be enumerated here and compared with cases in which more gradual pressure has been present.

One notes that the cortex as a whole was not narrowed, but was rather widened as result of an edema. The architecture was greatly disturbed, and especially so in the upper three layers. So great was the disturbance in the first, second, and third cortical layers, that in places no differentiation could be made between these. The first layer showed an excessive number of marginal glial cells, many of which were fibroblastic astrocytes, and within it were numerous fibroblastic astrocytes and macroglia, both of which were visible with the ordinary toluidin-blue stains, in alcohol fixed material. (Fig. 30.) Gitter cells were not visible. Small vessels were prominent. In the second layer the small ganglion cells were greatly reduced in number and glial cells were present in their places. In the third layer the amount of destruction of the ganglion cells reached its maximum, with enormous glial overgrowth.

The blood vessels in these layers stood out mainly because of swelling of the lining cells.

The ganglion cells in the third layer showed the picture that Spielmeyer has called ischemic cell disease, although other forms of cell disease are present.

The lower three layers likewise showed change of the same kind, but they were less intense than the upper three, but even here the ischemic cell picture was evident. The subcortex was completely overrun with macroglia cells with disintegration of the basic histologic picture.

With Cajal's gold-chloride stain, the enormous increase in fibroblastic macroglia was very evident (Fig. 31) and fairly uniformly distributed, but with certain important features. The astrocytes of the first and second cortical layers showed marked degenerative changes in the nature of clasmatodendrosis, those in the outer layer being almost undistinguishable, while those in the third layer were much better preserved, and in the lowermost three layers were hypertrophied and in good preservation.

There was great variability in the white matter; in places there were practically no macroglial cells. In the areas better preserved, there was an intense amount of well preserved glia, while intermediate areas showed broken up macroglial cells.

With Scharlach R. stains, one saw that in the cortex there was a slight increase in the lipid content, since practically every cell, ganglion and glia contained a little lipid. There was practically no fat about the vessels. The subcortex too contained more fat than was normal.

These findings can certainly not be interpreted as being due to the inflammatory reaction in the meninges, *per se*, but rather as a direct result of pressure on the blood supply to the cortex, by an intensely distended subarachnoid space, which had not been drained during life by lumbar puncture. It had occurred more acutely than from pressure by slowly growing lesion, but the changes are the same in character.

COMMENT.

In the course of our work on epilepsy, and the changes that occur in the brain and in the pachionian system, we were struck by the fact that cortical atrophy has been described so often in this condition, and has been seen by us on the operating table. We felt that the explanation given for the atrophy that occurs in arteriosclerotic and senile brains could not possibly hold for the younger and non-sclerotic cases, and so have made comparative studies as enumerated in this paper.

In the first place, we have studied the atrophic areas and have made actual comparisons with the non-atrophic areas in the same brain. We have also studied the brains of patients who have had pressure on the cortex by large, subdural hematomas, and also those in whom pressure from a meningeal inflammatory exudate occurred. We have been surprised to find that the changes in the atrophic portions of the cortex differ markedly from those areas of the brain that are free from atrophic changes of a gross nature. Briefly, it can be stated that in the atrophic areas there is a shrinkage of tissue with loss of ganglion cells, many of which show ischemic changes, and a marked increase of the fibroblastic macroglia, that at times approaches that seen in general paralysis of the insane.

In an attempt to get at the basis of these changes, comparative studies were made of cases in which cortical pressure had occurred

from outside sources. Here also the picture under the microscope was so similar to that found in the unexplained atrophic areas, and so different from that seen as a result of small and large areas of softening (thrombosis), that suggestion is made that the changes in the areas of gross atrophy seen in so many different conditions is primarily due to pressure of the overlying collection of fluid, rather than as an unexplained primary atrophy in which the cerebrospinal fluid assumes only a space-filling function, as suggested by Dandy.

In conjunction with the work that we have done on the pathology of the pacchionian granulations, we feel that there may be delay in the elimination of cerebrospinal fluid from the subarachnoid space, thus permitting excessive accumulations of fluid over the frontoparietal areas of the brain, with consequent pressure and gradual gross, focal, cerebral atrophy. Further study of this important phase of the problem is being undertaken. The findings fit in well with the knowledge that atrophic conditions of the brain occur most frequently in the region of the pacchionian granulations, at the vertex, and progressively extending along the pathways of cerebrospinal fluid circulation.

CONCLUSIONS.

(1) Encephalography has called attention to widespread areas of increased fluid collection and cortical atrophy, confined for the most part to the cerebrospinal fluid circulating pathways, over the cortex.

(2) A definite pathology of the pacchionian bodies has been established.

(3) In the light of the present accepted physiology of the pacchionian bodies, this pathology may predispose or give rise to impairment of cerebrospinal fluid elimination and consequent accumulations of fluid producing pressure along the course of its cortical pathways.

(4) Gross cortical atrophy which has been commonly recognized has been analyzed from the standpoint of vascular, tumor, inflammatory, and increased subarachnoid fluid accumulations, and a definite type of "pressure atrophy" has been differentiated from that arising in cases of cerebral vascular occlusion.

(5) Proof is offered that the atrophy probably results from a progressive vicious circle. (a) Pressure upon the cortex due to

gross lesions, as well as fluid accumulations. (b) Mild ischemic changes probably secondary to decrease in circulation. (c) Ganglion cell degeneration and disappearance associated with a fibroblastic glial increase. (d) Shrinkage of the cortex and subcortex consequent to pressure and degeneration.

(6) Comparison of microscopic sections from areas of the same brain, showing in the zones of atrophy, decrease in the number of ganglion cells and glial proliferation, as well as atrophic changes in the subcortex. These areas are usually found in the frontoparietal portions of the brain, whereas, the temporo-occipital regions are not involved in this atrophy, and sections show changes within normal limits.

(7) A possible hydraulic pressure mechanism responsible for the development of certain types of widespread atrophic degeneration of the brain is presented, and may be responsible for many types of gross changes noted, especially in the mentally defective, and chronic epileptic, or degenerative cases showing atrophy of unexplained origin.

BIBLIOGRAPHY.

1. Altman, Emil: Ueber die Umschriebene Gehernatrophie des Späteren Alters. *Zeitsch. f. d. gesamt. Neurol. u. Psych.*, B. LXXXIII, p. 610, 1923.
2. Bagley, C.: Functional Disturbances and Organic Alteration Following the Introduction of Blood into the Cerebrospinal Fluid. Paper read before Assoc. for Research in Nerv. and Ment. Diseases, December 28, 1927.
3. Bateman, J. Freemont: Closed Foramina of Luschka in the Brains of the Insane. *Arch. Neurol. and Psych.*, 14: 5, p. 616, 1925.
4. Cushing, H.: Studies on the Cerebrospinal Fluid. *Jour. of Med Research*, N. S., 26: 1, 1914-1915.
5. Cushing, H., and Weed, L. H.: Studies on the Cerebral Fluid and Its Pathway. *Bull. Johns Hopkins Hosp.*, 26: 367, 1915.
6. Dandy, W. E.: The Space Compensating Function of the Cerebrospinal Fluid. Its Connection with Cerebral Lesions in Epilepsy. *Bull. Johns Hopkins Hosp.*, 34: 245-251, August, 1923.
7. Dandy, W. E.: Impressions of the Pathology of Epilepsy from Operations. *Amer. J. Psych.*, 6: 519-522, January, 1927.
8. Dandy, W. E.: The Cause of So-Called Idiopathic Hydrocephalus. *Bull. Johns Hopkins Hosp.*, 32: 67-75, March, 1929.
9. Foerster, O.: Die Pathogenese des Epileptischen Krampfanfalles. *Deutsch. Zeit. f. Nervenhe.*, 94: 15-53, December, 1926.
10. Foerster, O.: *Zeitsch. f. d. ges. Neurol. u. Psych.*, 73: 4, 1921.

11. Fremont-Smith, F.: The Nature of the Cerebrospinal Fluid. *Arch. Neurol. and Psych.*, 17: 317-331, March, 1927.
12. Globus, J. H.: Glia Response in Chronic Vascular Disease of the Brain. *Arch. Neurol. and Psych.*, 20: 14, July, 1928.
13. Hassin, G. B.: Changes in the Brain in Increased Intracranial Pressure. *Arch. Neurol. and Psych.*, 20: 6, p. 1172, 1928.
14. Howe, H. S.: Physiological Mechanism for the Maintenance of Intracranial Pressure. Secretion and Absorption of the Cerebrospinal Fluid. The Relations of Variations in the Circulation. Presented before the Assoc. for Research in Nervous and Mental Disease, December 28, 1927.
15. Kennedy, Foster: Epilepsy and the Convulsive State. *Arch. Neurol. and Psych.*, 9: 567-575, May, 1923.
16. Kubie, Lawrence S.: Intracranial Pressure Changes During Forced Drainage of the Nervous System. *Arch. Neurol. and Psych.*, 16: 319-28, September, 1926. *Brain*, 51: 2: 244-253, 1928.
17. McCarthy, D. J.: Atrophy of Cortex in Tuberculosis. Fifth Annual Report of the Henry Phipps Institute. P. 225, February, 1908.
18. Pancoast, H. K., and Fay, Temple: Encephalography: The Roentgenological and Clinical Consideration for Its Use. *Amer. Jour. Roent. and Rad. Ther.*, May, 1929.
19. Weed, L. H.: The Cells of the Arachnoid. *Bull. Johns Hopkins Hosp.*, 31: 343-350, October, 1920.
20. Weed, L. H.: Hypotonic Solutions on Morphology. *Amer. Jour. of Anat.*, 32: 253, 1923.
21. Weed, L. H.: The Cerebrospinal Fluid. *Physio. Rev.*, 2: 171-203, April, 1922.
Experimental Studies of Intracranial Pressure. Assoc. for Research in Nerv. and Ment. Dis., December 28, 1927.
22. Weed, L. H., and Hughson, W.: The Cerebrospinal Fluid in Relation to the Bony Encasement of the Central Nervous System, as a Rigid Container. *Am. J. Physiol.*, 58: 85-100, November, 1921.
23. Weed, L. H., and Hughson, W.: Systemic Effects of the Intravenous Injection of Solutions of Various Concentrations, with Especial Reference to the Cerebrospinal Fluid. *Am. J. Physiol.*, 58: 53-84, November, 1921.
24. Weed, L. H., and Hughson, W.: Intracranial Venous Pressure and Cerebrospinal Fluid Pressure as Affected by the Intravenous Injections of Various Concentrations. *Am. J. Physiol.*, 58: 101-130, November, 1921.
25. Weed, L. H., and McKibben: Experimental Alteration of Brain Bulk. *Am. J. Physiol.*, 48: 531-558, May, 1919.
26. Weed, L. H., and McKibben: Pressure Changes in the Cerebrospinal Fluid Following Intravenous Injection of Solutions of Various Concentrations. *Am. J. Physiol.*, 48: 512-530, May, 1919.
27. Weed, L. H., and Wegeforth, P.: Experimental Irrigation of the Subarachnoid Space. *J. Pharm. and Exp. Therap.*, 13: 317-334, July, 1919.

28. Winkelman, N. W., and Fay, Temple: The Histology & Pathology of the Pacchionian System with Particular Reference to the Idiopathic and Symptomatic Convulsive States. *Arch. Neurol. and Psych.* 23: 44-64, (Jan.), 1930.

DISCUSSION

DR. ALBERT M. BARRETT (Ann Arbor, Mich.).—I wish that I were competent from personal experience to contribute something of value to the discussion of this interesting study by Drs. Fay and Winkelman, but unfortunately I can express little more than my personal appreciation of hearing this very important contribution.

Any extensive observation of brains from autopsies on psychotic patients impresses the observer by the frequent abnormal appearance which is found in the membranes and the diffuse varying degree of chronic atrophies of the brain hemispheres. These appearances are quite different from those usually seen in the autopsies of patients in general hospitals. These seem to occur without any of the vascular sclerotic or usual organic processes such as paresis or senility and are usually passed over as chronic meningitis or diffuse atrophies. It is by studies such as these that we will gain a better insight into the nature of these changes. What their relation may be to the distributions of mental functioning remains uncertain.

DR. ADOLF MEYER (Baltimore, Md.).—I am somewhat in a similar situation to that in which Dr. Barrett finds himself perhaps not so much from the point of view of my not being familiar with things of this sort under other conditions as because of my not being quite able to distinguish what is epilepsy and what is a general process observed also in other conditions. When I first came to Johns Hopkins and had an opportunity to see Cushing's operations, I was really startled by the regularity with which we found the damming up of cerebrospinal fluid over the cortex. That has always impressed me exceedingly strongly so that I quite agree with Dr. Fay that that is an exceedingly frequent and perhaps regular sort of an occurrence. I regret particularly the fact that Dr. Fay did not give us perhaps a few contrasting pictures of the disturbances with epilepsy and of the disturbances without epilepsy, with the findings of the Pacchionian granulations with epilepsy and outside of epilepsy. After all, we deal here with an exceedingly varying structure and for that reason ought to have very convincing examples of the various types of kindred disturbances and the correlation with epilepsy.

DR. TEMPLE FAY (Philadelphia, Pa.).—In closing this discussion, I just want to say that Dr. Meyer brought up the most important problem, but I have already transgressed upon your time. The actual differentiation between the various types and the epileptic will be presented by Dr. Winkelman before the American Neurological Society this June, and in this paper we also have the correlation which was impossible to bring out here. The films we showed you of the encephalograms are only partially those of epileptics. Some are post-traumatic, some idiopathic, some are senile types of gradual degeneration.



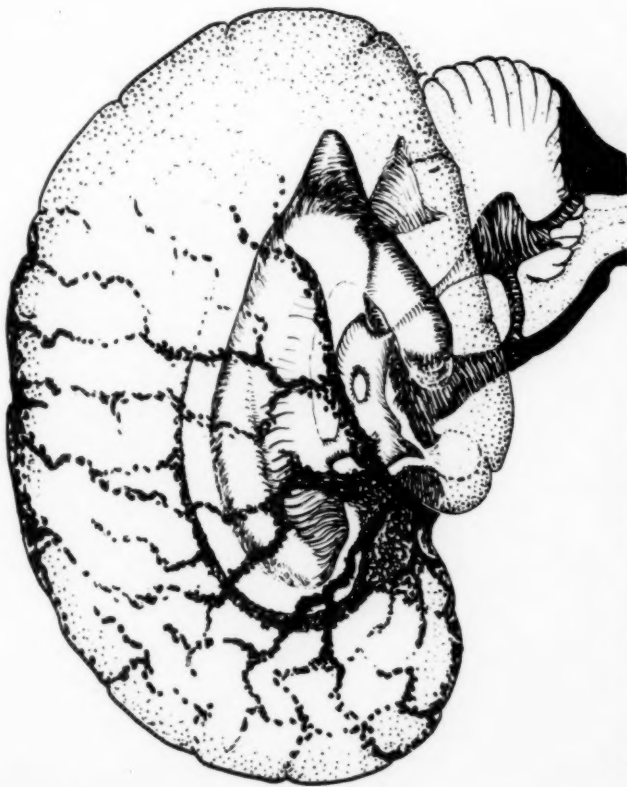


FIG. 1.—Reconstruction of the normal cerebrospinal fluid pathways. Note the underlying ventricular position with communication to the third ventricle, and thence by the aqueduct of Sylvius to the fourth ventricle. Fluid finds its escape through the foramina of Luschka, (lateral) and Megendie (midline posteriorly). It then becomes subarachnoid and passes into the cisterna magna and pons; thence anteriorly through the incisura tentorii, to reach the cisterna chiasmatis; from thence to be distributed over the cerebral hemispheres, and between the frontal lobes, to reach the pachionian bodies at the vertex, where the major portion of this fluid is eliminated. Note carefully the communication between the cisterna pons around the peduncle to the cisterna magna. The cisterna magna is situated over the upper surfaces of the cerebellum, just beneath the tentorium. Note the normal cortical circulating field does not include the temporal or occipital lobe.

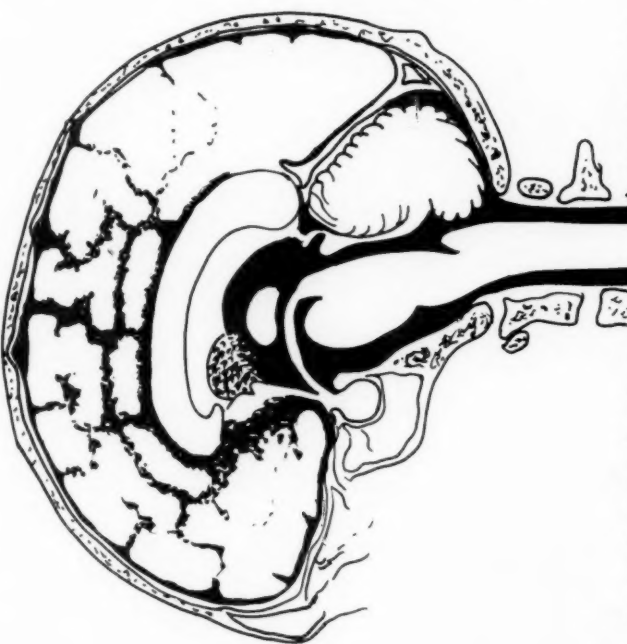


FIG. 2.—Sagittal section showing the cerebrospinal fluid pathways from the third ventricle to the pachionian bodies, by means of the mid-hemispheric route. The lateral ventricles are not shown. The situation of the pachionian bodies, at the vertex, usually three on each side, is indicated by slight indentations in the skull. Note freedom of the occipital lobe from the circulatory pathways, also the position of the cisterna magna. Magna Cerebri, where occasionally the pachionian body may be found.

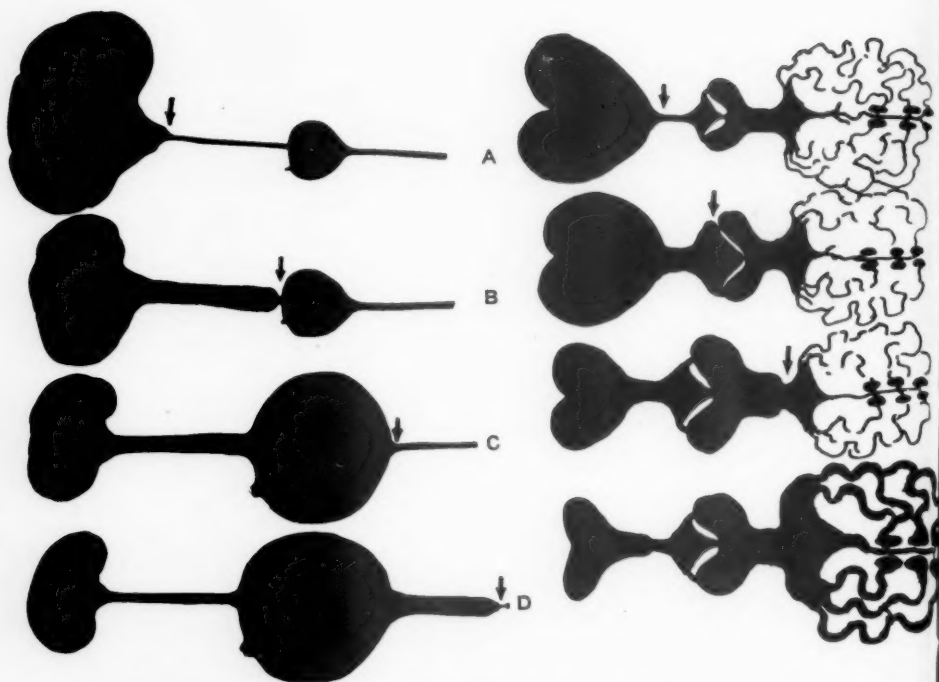


FIG. 3.—Diagrammatic comparison of the obstructions originating in the cerebrospinal circulating pathways and urinary tract obstruction. (a) Severe but incomplete obstruction of the hilum of the kidney produces hydronephrosis with dilatation and destruction (pressure atrophy). A similar dilatation in internal hydrocephalus is produced by an obstructive lesion at the opening of the aqueduct in the third ventricle. (b) An obstruction at the end of the ureter producing dilatation of the ureter and distal hydronephrosis. An obstruction in the fourth ventricle or at the Foramina of Luschka and Magendie produces a similar picture in that there is dilatation of the aqueduct, as well as the lateral and third ventricles, though not to as an extensive a degree as in "A." (c) Chronic obstruction at the neck of the bladder produces dilatation of the bladder, ureter (analogy complicated by Sphincter) and somewhat of the kidney hilum. A somewhat similar analogy exists in obstructions in the region of the cerebellopontine angle, or anterior to the Cisterna Chiasmatis, in that there is marked dilatation of the Cisterna Pontis and Magna; some dilatation of the fourth ventricle and aqueduct with moderate enlargement of the lateral ventricle. (d) Obstruction at the meatus produces dilatation of the urethra, bladder, and to a less extent to the ureter and hilum of the kidney, an obstruction at the outlet having a less immediate effect upon the structures at the source. Damage or obstruction in the region of the pacchionian granules produces dilatation of the cortical subarachnoid spaces; moderate dilatation of the cisternae, and some slight effect upon the ventricles. The changes represented vary in relation to the degree and location of the obstructing mechanism.



FIG. 4.—A normal encephalogram showing the size and position of the lateral ventricles, as well as the various cisternæ and cortical pathways.



FIG. 5.—Increase in cortical air markings over the frontal pole, with slight cortical atrophy.

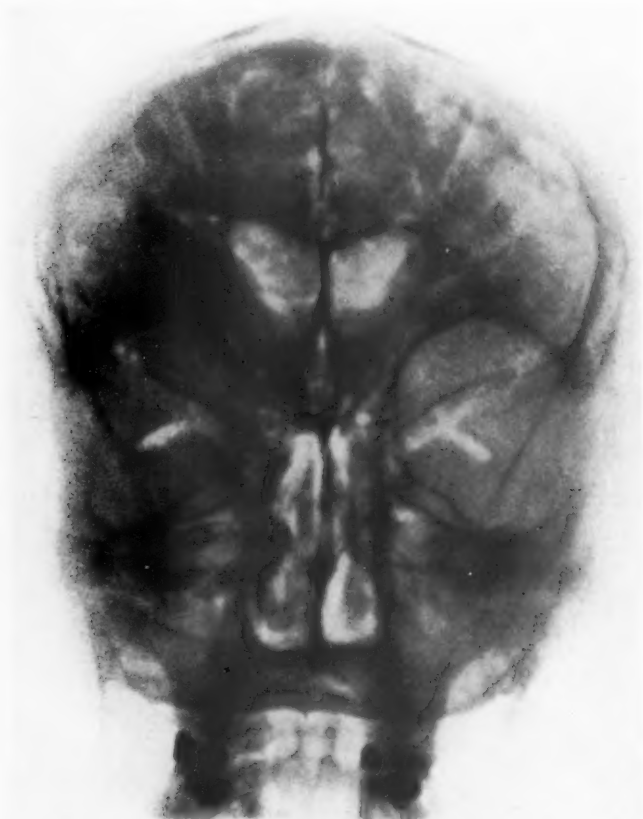


FIG. 6.—Widespread cortical atrophy with dilatation of fluid spaces over the frontoparietal areas.



FIG. 7.—Atrophy in the region of the pacchionian bodies, with enlargement of the Cisternæ Chiasmatis and Pontis, and slight dilatation of the ventricles.

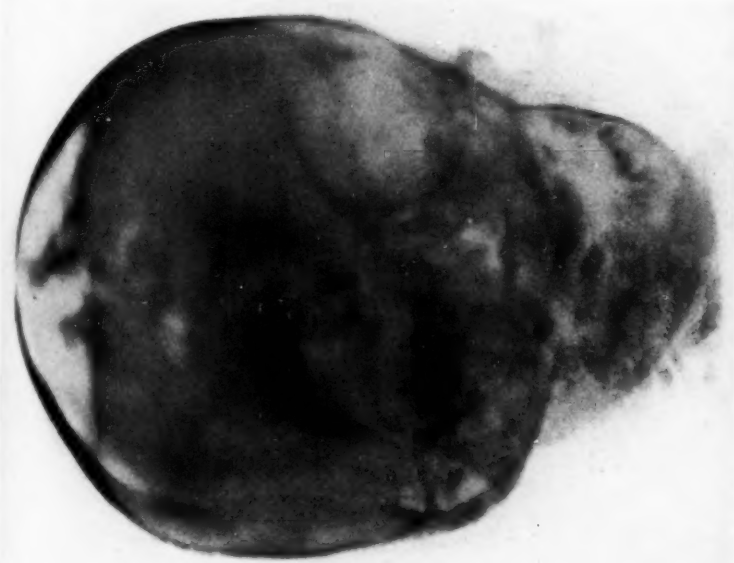


FIG. 9.—Bilateral cortical atrophy in a six weeks infant.



FIG. 8.—Marked, gross atrophy over the left cerebral hemisphere occurring in a three months infant. Note marked dilatation of the Cisterna Venæ Magnæ cerebri, extending over both cerebellar hemispheres.



FIG. 10.—Bilateral cortical atrophy extending between the hemispheres in a three months infant.

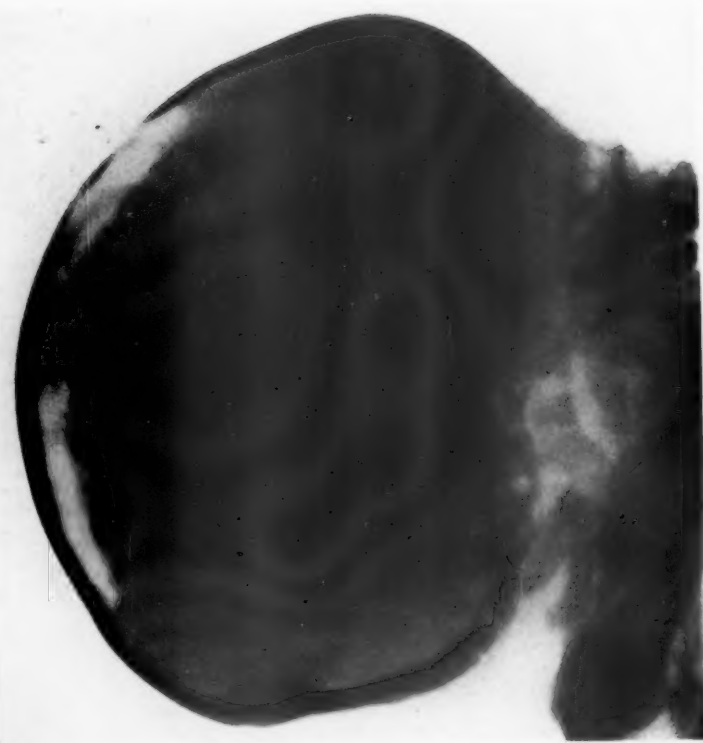


FIG. 9A.—Lateral view showing extent of the cortical atrophy and fluid level which indicates insufficient fluid drainage, and probably greater atrophy in the frontal areas than at the base.



FIG. 11.—Atrophy in the region of the frontal pole and the operculum in a three months infant.

VI.
in a three months infant
Fig. 9A.—Lateral view showing extent of the cortical atrophy and fluid level which indicates insufficient fluid drainage, and probably greater atrophy in the frontal areas than at the base.



FIG. 12.—Focal atrophy on the lateral aspects of the brain, of slight degree, with suggestive deformity of the right ventricle.

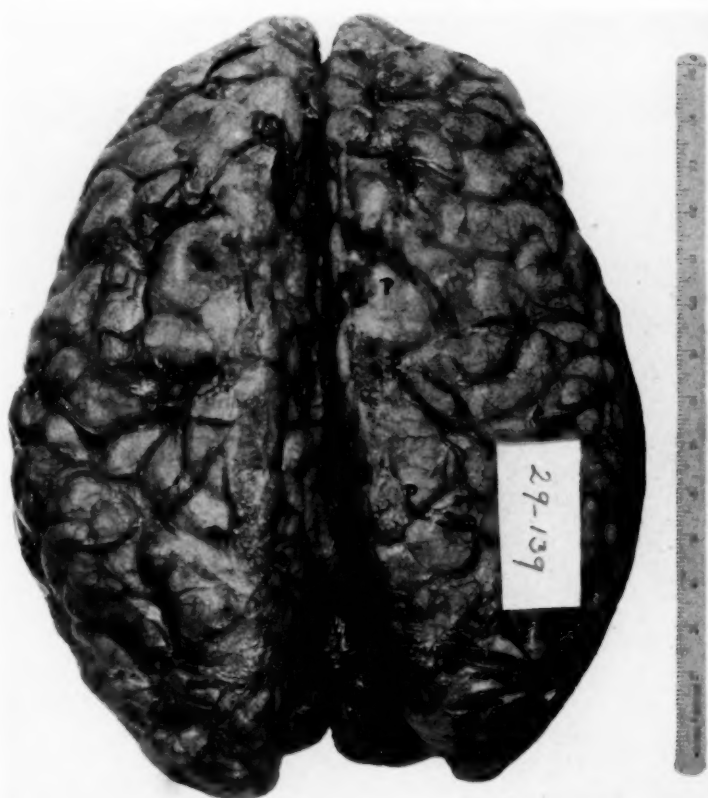


FIG. 13A.—Normal arrangement and appearance of pacchionian granules. (P.)



FIG. 13B.—Normal pacchionian system. Note pacchionian granulations at "P."



FIG. 14A.—Aplastic pachionian system. Note practically complete absence of all pachionian granulations. Adult epileptic patient.

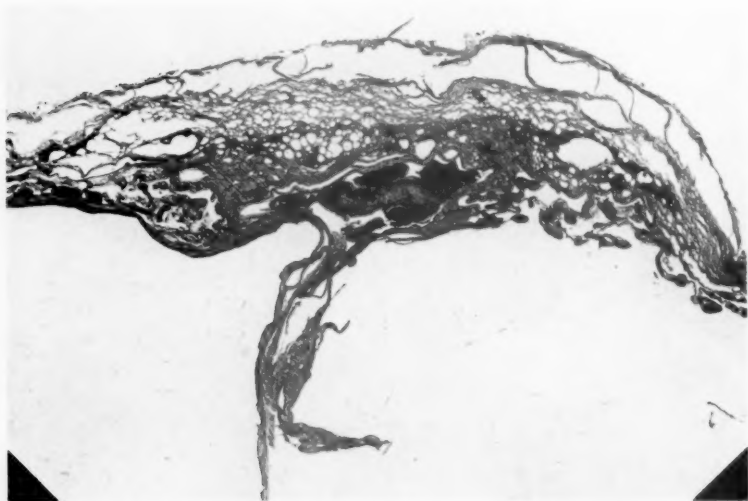


FIG. 14B.—Aplastic pachionian system (epileptic).



FIG. 15.—Aplastic arachnoid villus (A).

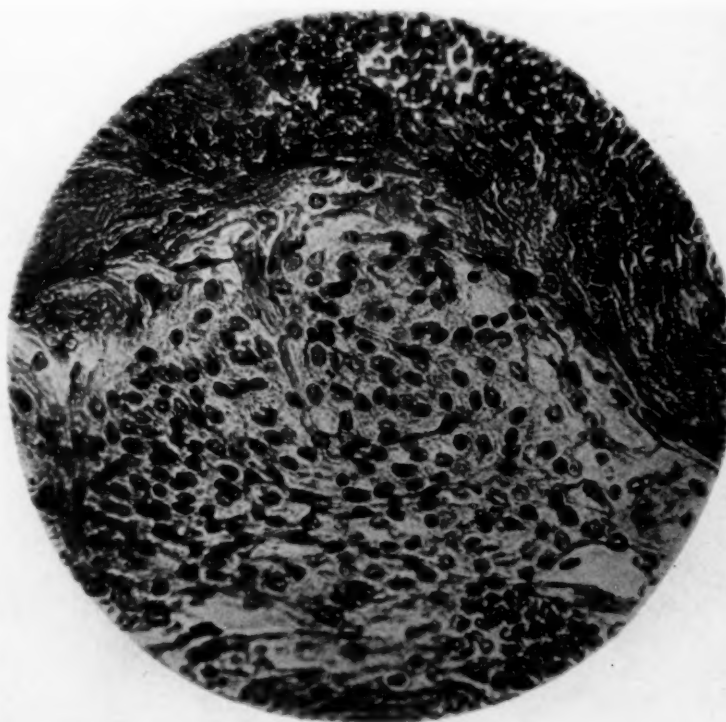


FIG. 16.—Compact grouping of mesothelial cells without formation of normal adult type of pacchionian nodule.

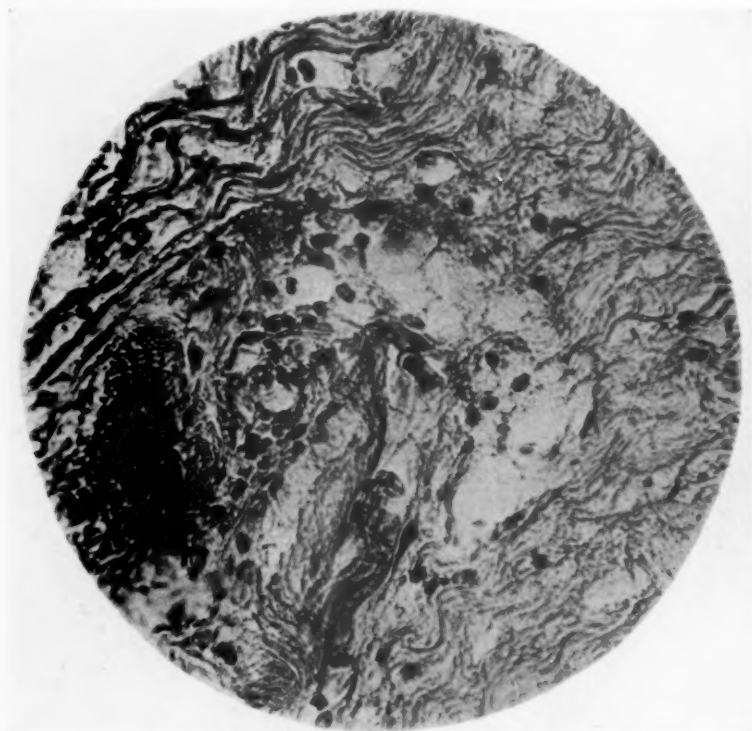


FIG. 17.—Edema of pacchionian granulation.



FIG. 18.—Fibrosis of pacchionian granule.

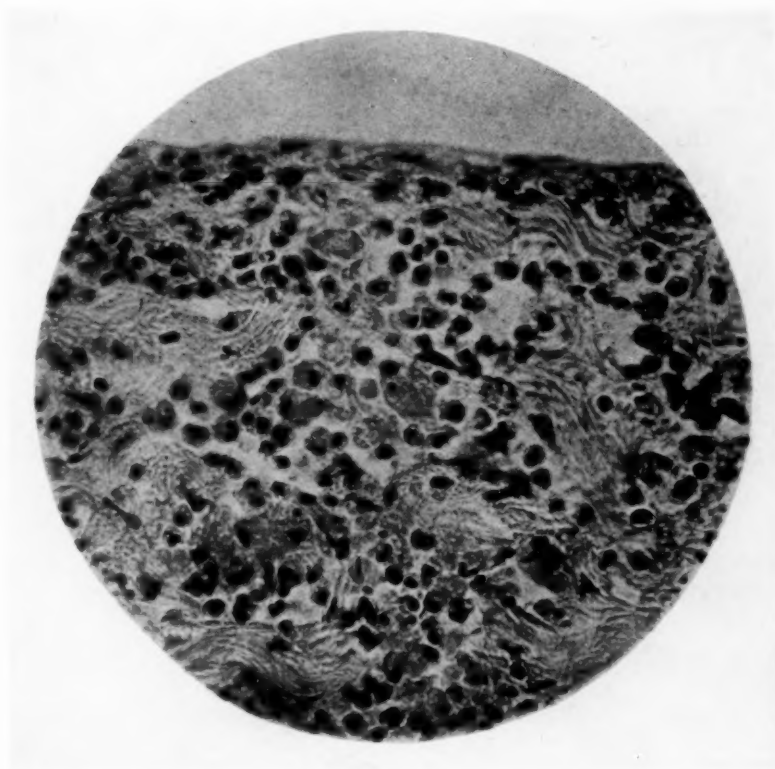


FIG. 19.—Acute inflammatory and phagocytic elements in the paccionian system.



FIG. 20.—Cortical atrophy, moderate, frontal area. (F.)



FIG. 21.—Cortical atrophy and pial fibrosis.



FIG. 22.—Gross appearance of softened area, *jaune plaque*. (S.)

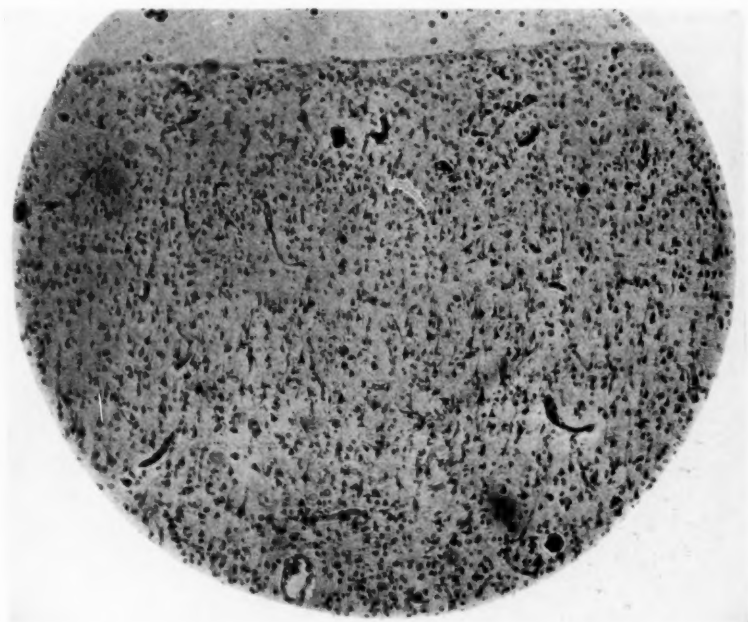


FIG. 23.—Cortical atrophic area. Note decrease in ganglion cells and difficulty of differentiating the individual layers. Toluidin blue stain.

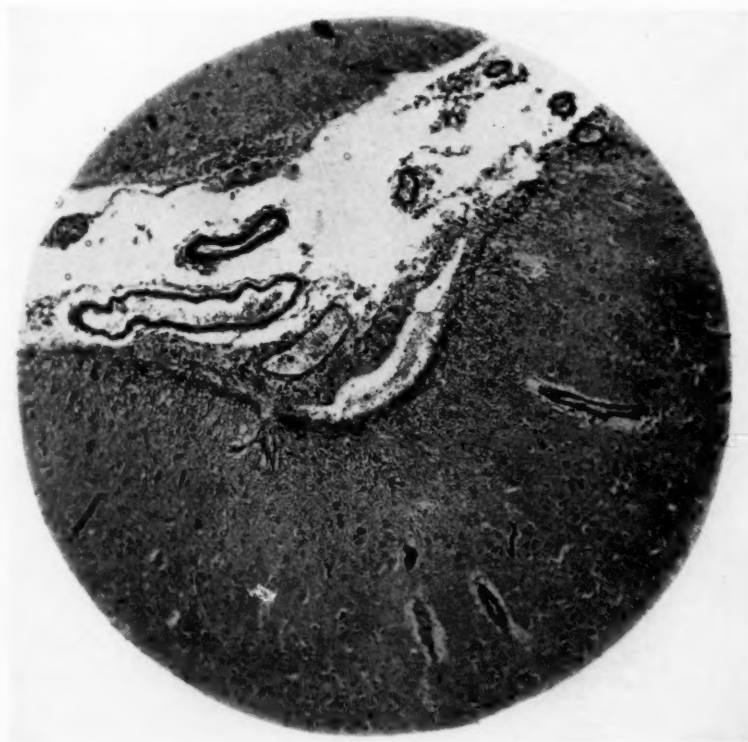


FIG. 24.—Marginal gliosis. (Phosphotungstic acid.)

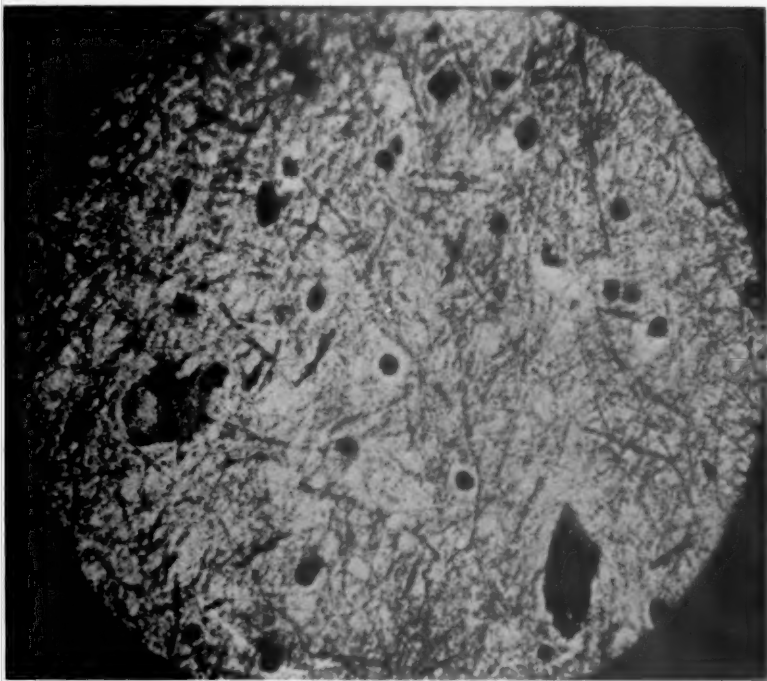


FIG. 25.—High power view of Cajal preparation of non-atrophic area (occipital) taken from same case as previous figure. Note practically complete absence of macroglial elements.

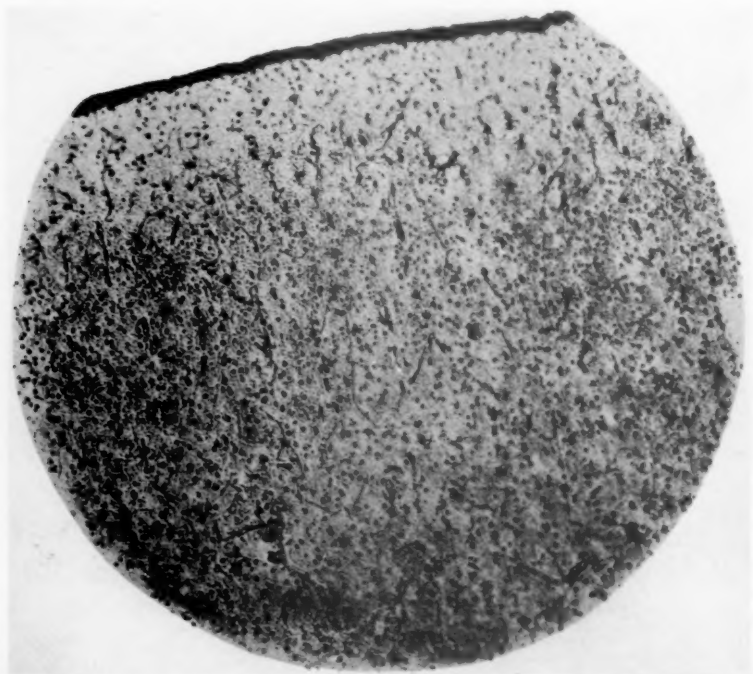


FIG. 26.—Marked macroglial increase in atrophic area of cortex. Cajal stain.

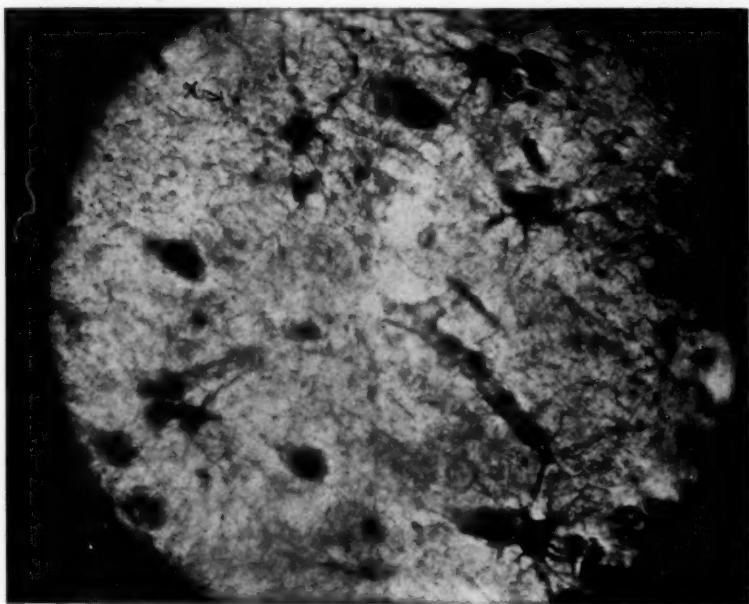


FIG. 27.—High power magnification of gray matter of atrophic area. (Frontal.)
Note clasmatedrosis. (x.)

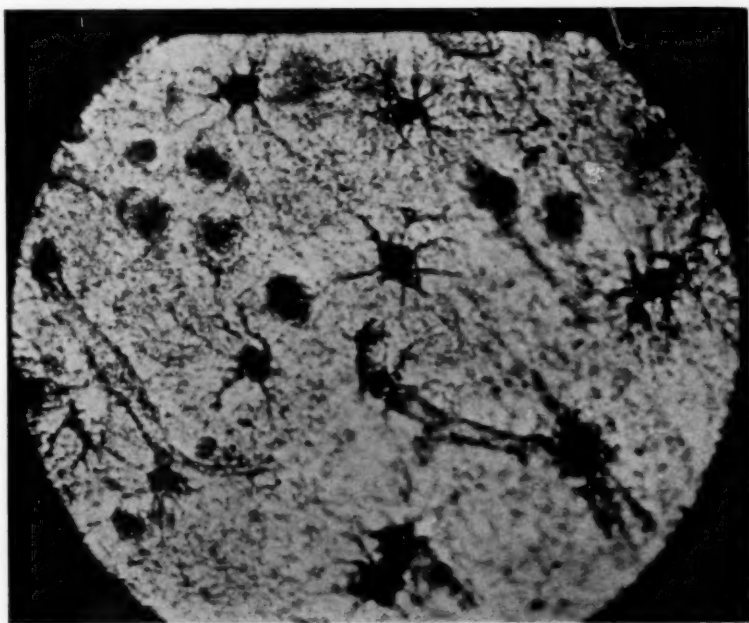


FIG. 28.—High magnification of white matter from atrophic area of brain.



FIG. 29.—Base of *jaune plaque*. (Scharlach R. stain.) Granular masses are glitter cells filled with red-staining lipid masses. Counterstained with Morgan stain. Note degeneration extending into white matter.

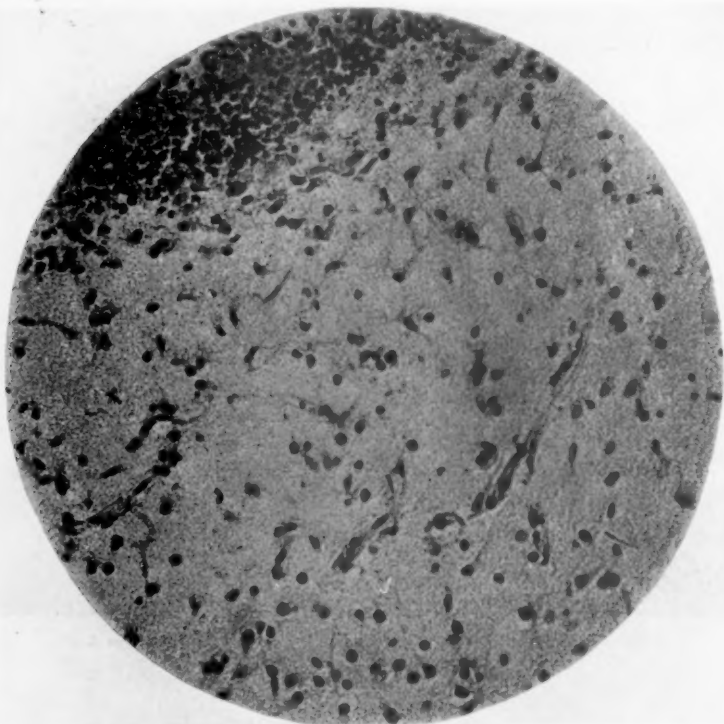


FIG. 30.—Marked glial proliferation of first cortical layer as result of meningeal accumulation of inflammatory elements.

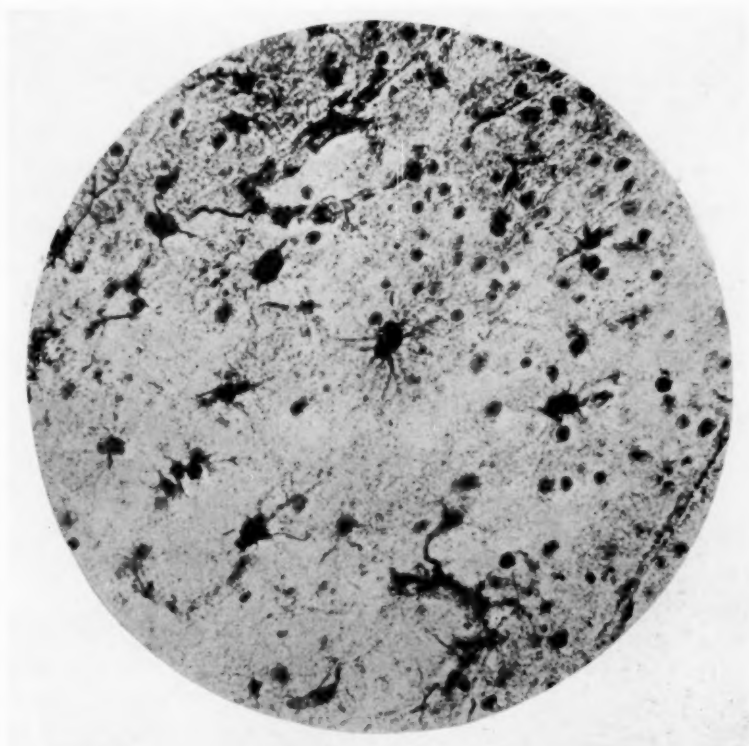


FIG. 31.—Cajal preparation of outer cortical layer in acute meningitis. Marked macroglial increase.

TH

T
bee
and
qui
and
opp
test

T
wo
tion
wa
to
If
line
me
hav
Th
sev
ma

Na
T
"Y
sta

—
*
As
Ne
cap
tro
2

THE RELATION OF EXTROVERSION-INTROVERSION TO INTELLIGENCE AND TUBERCULOSIS.*

By CLARENCE A. NEYMANN, A. B., M. D.

Tests for the determination of extroversion-introversion have been published by Rorschach, Travis, Laird, Woodworth, Pressy, and by Neymann and Kohlstedt. The last named test has been quite recently published.¹ Thus far it has not come into general use and since we believe that most of the members have not had an opportunity to apply it, we beg to give you a brief résumé of the test and its interpretation.

The test consists of 50 statements, each being followed by the words yes and no. As far as we were able to eliminate an implication of right or wrong in any of these statements, this viewpoint was excluded. The subject is asked to read these statements and to consider them from the standpoint of personal like or dislike. If the idea expressed is pleasing to the subject he is asked to draw a line under the word yes. If not under no. The following 50 statements serve as a key to the test. You will notice that 25 of them have the word yes underlined while 25 have the word no underlined. These key answers were obtained by applying the questions to several hundred individuals of known reaction types; namely, manic-depressives, schizophrenics, and normals.

THE NEYMANN-KOHLSTEDT DIAGNOSTIC TEST FOR INTROVERSION-EXTROVERSION.²

Name..... Occupation..... Age.....

This test is composed of 50 statements, each being followed by the words "Yes," and "No." There is no implication of right or wrong in any of the statements and you are asked to consider them from the viewpoint of personal

* Read at the eighty-fifth annual meeting of The American Psychiatric Association, Atlanta, Ga., May 14, 15, 16, 17, 1929. From the Department of Neuropsychiatry, Northwestern University Medical School, and The Chicago Municipal Tuberculosis Sanitarium.

¹ Neymann, C. A., and Kohlstedt, K. D., "A New Diagnostic Test for Introversion-Extroversion." *J. Abnormal and Social Psych.*, 23: 482-487.

² Distributed by C. H. Stoelting Co., Chicago, Ill.

like or *dislike*. Read the first statement and if you like the idea it expresses draw a line under "Yes." If you dislike it draw a line under "No." Proceed in the same way with the rest of the statements.

- | | | |
|---|-----|----|
| 1. Be by yourself a great deal..... | Yes | No |
| 2. Think of life in terms of pleasure..... | Yes | No |
| 3. Always be calm and collected..... | Yes | No |
| 4. Have a great deal of confidence in others..... | Yes | No |
| 5. Think or dream of what you will do five years from now..... | Yes | No |
| 6. Stay at home during a social affair..... | Yes | No |
| 7. Work with many people around you..... | Yes | No |
| 8. Do the same kind of work all the time..... | Yes | No |
| 9. Enjoy social gatherings just to be with people..... | Yes | No |
| 10. Think a great deal before deciding anything..... | Yes | No |
| 11. Accept suggestions rather than working them out for yourself.. | Yes | No |
| 12. Quiet rather than exciting amusements..... | Yes | No |
| 13. Dislike having people watch you..... | Yes | No |
| 14. Quit a tiresome task..... | Yes | No |
| 15. Save money rather than spend it..... | Yes | No |
| 16. Seldom (infrequently) analyze your thoughts or motives..... | Yes | No |
| 17. Indulge in reverie (day-dream) or thought..... | Yes | No |
| 18. Have people watch you do things that you do very well..... | Yes | No |
| 19. Let yourself go when angry..... | Yes | No |
| 20. Work better when people praise you..... | Yes | No |
| 21. Have excitement | Yes | No |
| 22. Often meditate and think about yourself..... | Yes | No |
| 23. Be a leader at a social affair..... | Yes | No |
| 24. Speak in public..... | Yes | No |
| 25. Do the things that you dream about (day-dream)..... | Yes | No |
| 26. Rewrite social letters..... | Yes | No |
| 27. Get things done very quickly rather than being slow and sure in
movement | Yes | No |
| 28. Think a great deal..... | Yes | No |
| 29. Be able to express your keenest feelings (joy, sorrow, anger,
etc.) | Yes | No |
| 30. Pay little attention to details..... | Yes | No |
| 31. Be exceedingly careful in meeting people..... | Yes | No |
| 32. Associate freely with people holding views opposed to your own. | Yes | No |
| 33. Puzzles | Yes | No |
| 34. Act on suggestions quickly rather than stopping to think..... | Yes | No |
| 35. Read about rather than do a thing..... | Yes | No |
| 36. Enjoy the story more than the way it is written..... | Yes | No |
| 37. Keep a personal diary..... | Yes | No |
| 38. Keep quiet when out in company..... | Yes | No |
| 39. Act on the spur of the moment..... | Yes | No |
| 40. Dislike thinking about yourself..... | Yes | No |
| 41. Always plan out work before you begin it..... | Yes | No |

- | | | |
|--|------------|----|
| 42. Change from one type of work to another frequently..... | <u>Yes</u> | No |
| 43. Avoid trouble rather than face it..... | <u>Yes</u> | No |
| 44. Believe that rumors are important..... | <u>Yes</u> | No |
| 45. Confide in others..... | <u>Yes</u> | No |
| 46. Distrust people you have just met until you get better acquainted. | <u>Yes</u> | No |
| 47. Study others rather than yourself..... | <u>Yes</u> | No |
| 48. Spend your vacation at some quiet place rather than at a lively resort | <u>Yes</u> | No |
| 49. Change your opinions easily even when formed..... | <u>Yes</u> | No |
| 50. Take an active part in all conversations going on around you.. | <u>Yes</u> | No |
| No. right No. wrong | | |
| Score = right minus wrong = | | |

Most normals are either outspoken extroverts or introverts. Scores between -10 and -20 and +10 and +20 predominate and make up more than 50 per cent of all individuals examined thus far. The remaining group make higher or lower scores while the neuroverts comprise about 17 per cent of the total.

It has come to be a generally accepted principle that high intelligence is an attribute of the introvert, the quiet individual with leanings toward autistic thinking. It was our problem to search out the truth of this premise on the basis of actual test material and actual comparisons. Intelligence can be measured so accurately by the alpha test of Yerkes and Yocum when large groups are considered, that the former difficulties of presumptive evidence can be definitely overcome. We believe that our test in a large measure excludes the intricacies and complications, as well as much of the error, of the work of Rorschach, Pressy, Woodworth, and others; that an accurate estimation of degree of extroversion-introversion can be obtained through its use. It has also been a general presumption among physicians that patients suffering from pulmonary tuberculosis in its various manifestations were happy, cheerful, and if anything rather elated individuals—therefore of an extrovertive type. This was the other phase which interested us.

We selected 300 patients of the Chicago Municipal Tuberculosis Sanitarium, 300 students of the college of liberal arts of Northwestern University, and 300 factory workers as our subjects. In order to avoid any question of sex, half the individuals chosen in the first 600 were male, half female. The sampling was entirely at random except that we divided the patients of the sanitarium into

two groups. Those suffering from minimal tuberculosis belonging to the minimal A group, and those suffering from advanced pulmonary tuberculosis. The first were inmates of the cottages, had no fever over long periods of time, and for all practical purposes can be considered as semi-ambulant cases. The second group was confined to bed with more or less intense symptoms. Three hundred patients and 300 college students were given the alpha test and the Neymann-Kohlstedt test. It would seem almost unnecessary to state that the intelligence of these groups as well as their social position showed an extreme variation. The college students belonged predominantly to the A intelligence group. There are only about 20 per cent of B and 3 per cent of C intelligences in this group. The sanitarium group, on the other hand, showed:

9 per cent A	intelligences.
14 per cent B	intelligences.
28 per cent C +	intelligences.
23 per cent C	intelligences.
14 per cent C -	intelligences.
7 per cent D	intelligences.
5 per cent D -	intelligences.

The last two groups, only 12 per cent of the patients, belong to the definitely feeble-minded classification. In other words, the average intelligence at the sanitarium is higher than that given by Yerkes in his examination of 1,700,000 men of the draft. His figures being:

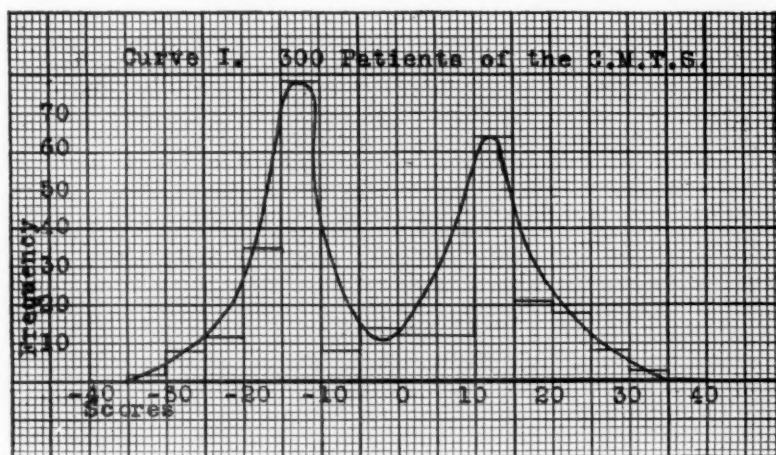
4½ per cent A	intelligences.
10 per cent B	intelligences.
15 per cent C +	intelligences.
25 per cent C	intelligences.
20 per cent C -	intelligences.
15 per cent D	intelligences.
10 per cent D -	intelligences.

Our scores are accounted for by the fact that the Municipal Tuberculosis Sanitarium will not accept the obviously feeble-minded and Chicago, being an outstanding industrial center, naturally has a smaller percentage of non-institutionalized mental defectives than the country as a whole. We therefore believe that this sample of the patients at the sanitarium, who are admitted from all classes of society and are not necessarily indigent paupers, gives us

a fair representation of the intelligence of the city at large. The group of college students, as mentioned previously, represents an exceedingly selective group.

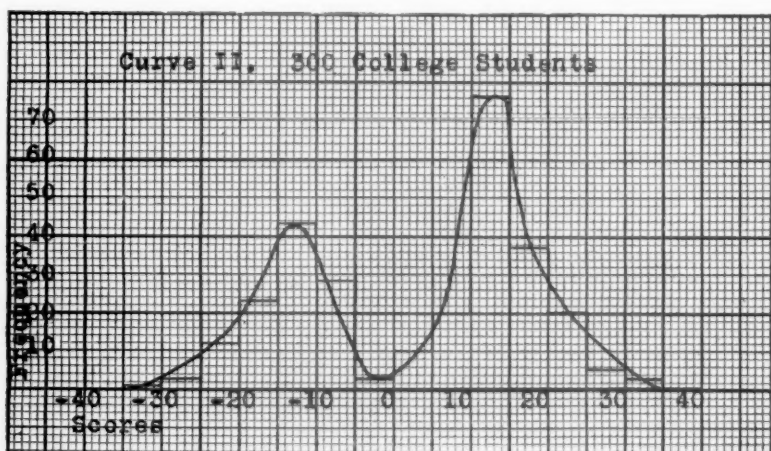
Plotting the extrovert-introvert traits of all sanitarium patients gives us the curve shown in Curve I.

A high incidence of introverts is shown, amounting to 46 per cent in all, a somewhat lower number of extroverts, namely 39 per cent, and 15 per cent of neutroverts. The introvertive peak lies between the scores of -10 and -15 . The extrovertive peak lies between the scores of $+10$ and $+15$. In other words, the introverts slightly



exceed the extroverts. Furthermore, the cases of minimal tuberculosis show a greater predominance of introversion. The percentage here being 48 introverts and 36 per cent extroverts while the more advanced cases, 80 of the 300 patients show a leaning toward extroversion. Here the results are 41 per cent introverts and 47 per cent extroverts. Perhaps their long confinement, for most of these cases had been sick for months and even years, had some bearing on their desire to go out and have a good time; a purely extrovertive trait. Without setting this up as a hard and fast standard, we may therefore conclude that there is a decided tendency towards extroversion as tuberculosis progresses and the patient becomes bed-ridden. Introversion, however, is more prevalent in the entire group.

The college group shown in Curve II manifests a decided leaning toward extroversion. Here we find that 48 per cent of the group classify as extroverts, 32 per cent as introverts, and 20 per cent as neutroverts. One is at first prone to assume that, contrary to popular opinion, group extroversion and group intelligence have a tendency to go hand in hand. We cannot be absolutely sure of this factor however, because college youth is prone to progress with a certain amount of dash and don't care attitude and a premium is placed on push and initiative in our modern universities. The patients at the sanitarium are, for the most part, young individuals,

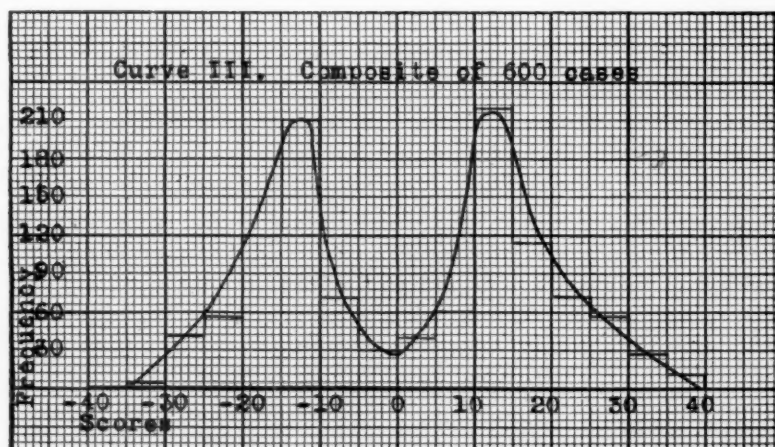


but the general age exceeds that of the college students. Age tempers types and difficulties in life probably have a tendency to make the personality more quiet, sedate, and thoughtful. We can be sure of one thing at least, that though extroversion need not necessarily go hand in hand with intelligence, introvertive traits are associated with this factor to no greater degree. The premise that great thinkers or groups of the intelligentsia belong predominantly to the introvertive type must therefore fall by the wayside.

Finally we can show a composite curve (Curve III) which presents both groups.

This curve shows results from the examination of 600 individuals. It shows beyond a doubt that aside from all factors of intelligence, environment, social position, etc., society generally is divided

into two great groups of about equal size; 40 per cent introverts and 40 per cent extroverts. A lesser group of 20 per cent, the neutroverts, exist either as a separate group or as a composite picture of other groupings. The factory workers showed a somewhat higher composite intelligence level than the hospital group and of course a much lower intelligence level than the college group. Workers in industrial plants are suspicious of the psychiatrist and the testing had to be done by simply passing the forms around and asking individuals to mark them during their spare moments. The alpha rating had been obtained when they first applied for work,



in some cases several years before this time. Under these conditions, the number of neutroverts rose to about 39 per cent. This is due to the fact that introverts are suspicious and cannot seem to make up their minds how some of the questions should be answered. Therefore they neglect to answer them or underline both yes and no answers. Thus they fall into the neutrovert group with ratings between -6 and $+6$ accepted as a standard for this group. The curve obtained approaches a smoothed curve under such unfavorable conditions. In the other two classes, the college students and sanitarium patients, neutroverts were given individually supervised tests, reducing the original 25 per cent to about 17 per cent. The same comparative high incidence of introversion with the less intelligent group of factory workers was demonstrated.

We have thus far examined about 1300 individuals. The reports on 400 were published in our preliminary paper and included many outspoken psychopathic cases. Besides these, about 25 border-line cases have been examined. Here the diagnosis between schizophrenia and manic-depressive insanity was in doubt. The outcome of further clinical study on these cases has shown that practically all of them closely agreed with the scores obtained by the test. Those with outspoken introvert scores were finally grouped as schizophrenics, while those with outspoken extrovert scores were grouped for the most part as manic-depressives and showed a much higher recovery rate than the first group.

I wish to thank Mr. Kenneth D. Kohlstedt for his assistance in grading the college students as well as in preparing the curves.

SUMMARY.

1. Nine hundred normal individuals have been examined by the Neymann-Kohlstedt test.
2. Most individuals fall into one of the two groups, the introverts and the extroverts.
3. Intelligence and introversion do not coincide.
4. The average tuberculosis patient has a strong leaning toward introvertive qualities.
5. The introvertive qualities decrease as pulmonary tuberculosis becomes progressive and the patient becomes bed-ridden.

DISCUSSION.

DR. A. A. BRILL (New York, N. Y.).—An undertaking like this is very interesting. I believe it is a very ambitious one, perhaps a little too ambitious. To say, for instance, that normals are either outspoken extroverts or introverts, I believe, is a bit daring. We must remember that the individual, if you follow him through life, is usually extroverted in childhood. It is the exception to find an introverted child and does not betoken well for the future. As the individual grows older he usually becomes more and more introverted. Even the manic patient who is the classical extrovert, is not always so when he is depressed or in a depressive mood—he is preponderantly introverted. We all know that there are schizoid manics who are now introverts and now extroverts, depending on when they are examined. In other words, I believe that it is very difficult to say whether a patient is definitely this or that. With Bleuler, we can say that it is simply a question of to what extent one is an extrovert or syntonik personality, or an introvert or schizoid personality. Let us take as an example the doctor's own conclusion about tubercular patients, particularly those who have run a chronic course. Dr. Neymann tells us that they are preponderantly extroverted and that is un-

doubtedly true if one forgets that chronic tubercular patients show the so-called *speciosa* so well described for centuries. On the surface they seem extroverted, but deeper studies show that these patients know that they are in a bad, precarious condition, repress it, and then react with euphoria. But this reaction does not show that we are dealing with extroverts exclusively. We know these reactions are purely superficial and often change with time, with disease, and with age. Such a test should not be taken in the definite and almost mathematical formulations given by Dr. Neymann.

DR. WILLIAM A. WHITE (Washington, D. C.).—Mr. President, I suppose it is asking a good deal to expect from Dr. Neymann that he should prove by any thesis that the introvert is superior to the extrovert, because I take it, if Kretschmer is correct and the introvert is predominantly asthenic and the extrovert pyknic, that the general appearance of the reader will indicate a prejudice perhaps in the conclusion.

I want to voice my agreement with what Dr. Brill said in his criticism and to comment upon some of the results which we have had at St. Elizabeths Hospital, in terminal situations, after the story has all been told, the tissue changes laid down and the autopsy has been performed. Preponderantly we find tubercular lesions in the schizoid group: they die of tuberculosis in a very large percentage of cases. Preponderantly we find that the manic-depressive group has a minimum of tubercular lesions and die infrequently of tuberculosis. These conclusions must be taken as statistical conclusions; they don't necessarily apply to any particular individual. So when Dr. Neymann was giving his conclusions about the preponderance of extroversion in advanced tuberculosis, it seemed to me that that was just the opposite of what we found, but as I followed his paper I wondered if it wasn't perhaps confirmatory to a certain extent in some such way as Dr. Brill has indicated. I have formulated the distinction in some such way as this, that the schizoid make-up presents, when he gets a psychosis, a decompensating type of psychosis which is associated when he has a somatosis with a decompensating somatosis, and *vice versa* with the manic-depressive group. Therefore, I am wondering whether the advent of extroversion in a chronic tuberculosis may not be an expression at the psychologic level of the attempt of the organism to effect a cure. It does seem to us that the type of pulmonary tubercular patient who has his tuberculosis over a long period of years, who develops a fibrous protective mechanism and who tends to go on living indefinitely and not dying of his tuberculosis, is pretty apt to be an extroverted type of person, speaking again statistically. So it would seem to me perhaps somewhat more important, from my point of view to ask the question, not whether Smith or Jones who has tuberculosis of an A or a B or a C degree, is at that particular moment extroverted or introverted, but whether Smith or Jones at any particular time is presenting processes which are compensatory, protective and curative, or decompensatory and destructive, and whether associated with those processes there may be extroversion or introversion. If Dr. Neymann can answer that question in his conclusion, I wish he would.

PRESIDENT ORTON.—I am glad Dr. White, in order to leave a path of escape for the pyknic habitus on a statistical basis, also made it possible for us of the opposite or asthenic type, to escape. Is there further discussion? If not, I will ask Dr. Neymann to respond.

DR. CLARENCE A. NEYMANN (Chicago, Ill.).—I am afraid that Dr. White's question is too difficult for me to answer.

The comments of Dr. Brill were very apt and to the point. I mentioned, if Dr. Brill will recollect, that the type does change and that as the individual grows older it is likely to become modified. With this in mind about 25 business executives of Chicago were examined. These were true leaders in the community. It was astonishing to note that many executives who were considered introvertive by me in my personal association with them, really proved extrovertive in type. Their attitude while answering this series of 50 questions was about as follows: "Yes, Doctor, I should do this, therefore, I would do it, but my inner tendency and feeling is to do the opposite." In such cases this tendency and feeling was taken as the criterion for the test; the attitude, a product of experience and adjustment, was discounted.

It is too early to give definite data about this change in type due to experience and adjustment towards life. We are anxious to ascertain whether this test in the hands of others will give approximately the same results that we obtained.

THE LANGUAGE OF THE PSYCHOSES.*

By WILLIAM A. WHITE,

St. Elizabeths Hospital, Washington, D. C.

More or less coincident with the assumption by medicine of something approaching a scientific attitude, as it emerged from the realms of mysticism, there was an attempt upon the part of the doctor to find out something about the human body, something of its structure and something of its function. In the study of its structure and function we have developed the broad basic sciences on which medicine is founded, anatomy and physiology. Both of these fields of inquiry were to begin with largely speculative. Structure, however, was visible and tangible and it needed only the moral courage to dissect in order to find out something definite about it. So the early physicians realized the importance of knowing about the body and as medicine developed this importance became progressively acute until those things that could be simply observed were supplemented following the invention of the microscope with those things that needed its aid. The various tissues and organs of the body have in the course of the years been subjected to careful inquiry as to their structures and as to their functions; and parts of the body that were not ordinarily at first considered as either tissues or organs have also come under this survey, such as the fluid parts of the body, the blood, the lymph, the cerebro-spinal fluid.

All of this information so gathered we might, by analogy or by a perfectly proper extension of the meaning of the word, call the language of the body. Speaking after this fashion, the area of cardiac dulness, the frequency of the heart beat, the sounds disclosed on auscultation of the heart, might be spoken of as the language of the heart; and to carry the analogy still further, when the various organs are diseased the signs and symptoms which they develop might be spoken of as the language of disease. This is an extension of the term, however, which I shall not indulge in

* Read at the eighty-fifth annual meeting of The American Psychiatric Association, Atlanta, Ga., May 14, 15, 16, 17, 1929.

here, for to write on the language of the body and the language of disease from this point of view would involve a complete treatise on the structure and the functions and the pathology of the body.

There is a broad sense, however, in which the word "language" is used which should be mentioned, and that is on the theory that language is a means of communication and that what one person learns from another is learned through the medium of what may be termed language. Not only the spoken and the written word are included, but facial expressions and bodily postures are included; and of course if one wishes to pursue this matter still further, particularly by the investigation of the emotions, he would have to include visceral postures as well, such as the increased tonicity of certain parts of the gastro-intestinal tract under conditions of anxiety, increased blood pressure under certain emotional conditions, the rapidity of the heart action resulting from fear, and a thousand other things.

In this paper, however, I shall not consider these widely flung territories which by analogy might be brought under the concept of language. I shall include primarily only the written and the spoken language, with the addition necessarily of the various intonations, modulations of the voice, and the expressions which go with the spoken word and help to give it meaning, and certain gestures and particularly drawings which help so materially in understanding what is going on in the mind of the subject. In other words, I shall consider only those aspects of language with which we are more or less familiar and which give us fairly definite and first hand information about the state of mind of the subject. And in doing so I wish to follow out the analogies with which I have begun and indicate to you that it is my belief that the examination of language as such, its form and substance, structure and function, is on all fours with the examination of a tissue or an organ, and from the point of view of its importance is much more highly significant for the psychologist or the psychiatrist than any examination of a tissue or an organ can possibly be. In fact language, by analogy, and here I speak in the restricted sense of the spoken and written language in the main, may be considered as an organ; and if we were to define its most general purposes as we define the purposes of what is more commonly known as an organ, if we were to strive to formulate its main

objective as we might the liver for example by saying that the main objective of the liver is the handling of carbohydrate metabolism, we should then say that the main function of language was as the most important tool that man possesses with which to modify the environment to suit his purposes and his needs. When we realize that language is an expression of man at the psychological level, that it is not simply an expression of certain mechanisms of the larynx, the tongue, the muscles about the mouth and certain resonances depending upon the size and the shape of the oral and nasal cavities and the adjacent sinuses in addition to the tension of the vocal cords, etc.—when we realize that language is not such a physiological function of any particular part of the organism but is an expression of the organism-as-a-whole, that is, at the psychological level, and that it is through the medium of language that we get our most valuable information of what is going on at this psychological level, its supreme importance for psychology and psychiatry and for psychopathology and psychoanalysis becomes at once apparent, as does its preponderant importance over and above that of any other organ, I might say, the brain not excepted.

A recent statement by Millikan, made at a gathering of eminent scientists, arrested my attention and seems to give expression to the tremendous significance of language as disclosing man's psychological processes. He said:

In the last analysis there is nothing that is practically important at all except our ideas, our group of concepts about the nature of the world and our place in it, for out of these spring all our conduct.

This seems to me to be a variant upon the well-known saying of Heraclitus that "Man is the measure of all things," and with the establishment of the new Institute for Human Relations at Yale University we appear to be coming into a recognition of these profound truths.

Before proceeding to the matter of my essay in further detail let me read one further quotation, from a German philologist, which illustrates the significance of language in a general way so clearly that you will be prepared to follow it in somewhat greater detail. He says:

In language the whole intellectual and moral essence of a man is to some extent revealed. "Speak, and you are" is rightly said by the Oriental. The language of the natural man is savage and rude, that of the cultured man is

elegant and polished. As the Greek was subtle in thought and sensuously refined in feeling—as the Roman was serious and practical rather than speculative—as the Frenchman is popular and sociable—as the Briton is profound and the German philosophic—so are also the languages of each of these nations.*

I have chosen the subject of my paper, first, because I believe that the results of an examination of the language of our patients, both as to its grosser and its microscopic elements so to speak, would result in the writing of a new chapter in psychiatry and, secondly, because I think that there is now enough material that has been gathered regarding the development of language among savages and in children and its various disturbances, as the result of cerebral lesions particularly, to warrant at least an effort to begin the writing of this chapter. I think, further, that the study of the language of our patients is of peculiar significance and importance. In the first place, it gives us at once and first hand certain information which is otherwise gained only in a roundabout way and in an indirect fashion, and, secondly, I believe we have in language the very best approach to that problem that has so vexed us for so long—the problem of the distinction and the relations between the organic and the functional, because language expresses within itself changes of either functional or organic origin and so these two aspects of the organism find their expression through its single medium.

In approaching the problem of language in somewhat more detail, the first thing that I will call your attention to and one which I think to be of outstanding significance is that there has gradually come to pass a change in our ideas of the development of language which is along the same lines as the change in our ideas in regard to the development of the mind or of the development of the body, or for that matter of anything living. We used to think of the mind as if it grew by a process of addition. We thought of the psychological units as so many sensations which somehow or other added together produced a perception. The sensation of yellow, of roundness, of a certain degree of consistency, of a specific type of odor and taste, were somehow put together with the result that we perceive an orange. We know now in the field of psychology, as we do in the field of philology, that this is not so, and yet we still

* Jenisch, D.

act as if it were so in certain fields of endeavor. I cannot refrain from saying that we have not as physicians yet learned that we cannot add together a urine analysis, a blood count, a metabolism report, the results of a Wassermann test, an X-ray of the sinuses, and all the rest of the hundred things that the different specialists may report upon—we cannot, I say, add the reports of all these specialists together and in that way make or discover a human being. The personality, the character, everything that makes an individual significant for us who know him, escapes any such method of procedure. Again I emphasize the importance of language, because with the necessity of understanding the language of the patient, with the necessity of listening to what he has to say and evaluating it, there would necessarily come into the picture some, at least, of those very things which are left out by the methods now so frequently pursued.

This change to which I refer is a change away from the concepts of what has been called the old "faculty psychology" in virtue of which the individual was supposed to operate because of the possession of such faculties as the intellect, the will, each of which operated somewhat as a *deus ex machina* and brought to pass certain results by playing upon the mind and the brain of the individual. The dynamic concepts of the present century, included in this particular instance by the Gestalt or Struktur psychology, have shown us how far wrong we were in such simplistic methods of approach. The term "organism-as-a-whole" really means something, and it means that the organism in all its various parts, including the mind, works as an integrating mechanism each part of which is related to every other part. So that there is no place in such a scheme for a faculty, which is somehow outside the circle of influence of the organism. As an illustration of my meaning there occurs to me the bright idea of a boyhood playmate of mine, who conceived the possibility of learning a language on the principle of learning one new word every day. At the end of his year his vocabulary would then consist of three hundred and sixty-five words, which would be sufficient at least to express thought in its simpler forms. This is a fair idea of the concept of the growth of the mind by a process of addition, as manifestly impossible as to learn a language by any such method as that above described and which leaves out of consideration the grammar of the language, the significance

of prefixes and suffixes, the qualifying words and the mechanism of sentence formation, dependent and independent clauses and the whole problem of the relationship of words and groups of words to one another. In the same way the additive theory of language leaves out of consideration the whole question of relationship, of synthesis.

On the basis of this additive theory of language schools in my day taught us how to read and write by first teaching us the separate letters of the alphabet, then combining the alphabet into small words of two or three letters, then combining the words into short sentences. This method has now been superseded, at least in many places, because it is not the method by which one naturally learns to talk. The first word that a child learns all over the world pretty nearly is the word "ma-ma." Stern says that the word ma-ma, or a similar sound meaning mother, has been found in no less than one hundred African languages. The significant thing, however, to which I wish to call attention is that this first word ma-ma is not a word at all, but in the sense of the Gestalt psychology and in reality it is a sentence. No mother has any doubt of this fact in her attitude toward her child, for the word ma-ma may be uttered by the child in innumerable ways depending upon the situation, on the inflection, on the association with additional sounds or cries, etc., so that it may mean any one of numerous things, such as a cry for the mother to come here, an indication that the child is hungry or that the child is suffering some discomfort or is frightened or what not, and all these various meanings the mother interprets with perfect ease, knowing exactly what the child's cry of ma-ma at that particular time and under those particular circumstances means. The subsequent development of language, then, is not along the lines of the additive scheme previously outlined, but is by a parallel process of analysis and synthesis. What is acquired first is not a letter of the alphabet but a complete sentence, and so in reality the development occurs in precisely the reverse direction, from sentence to word and from word to letter.

An appreciation of this change from the old additive theory of the growth of the mind with its emphasis upon association and the present Gestalt idea, which sees in the simplest perceptions a form of synthesis, is fundamental to an understanding of language, whether the language of the normal individual or of the psychotic.

The child when it first opens its eyes may and undoubtedly does see a world very different from the world we as adults look upon, but it is not a world made up of unit sensations which subsequently flow together into perceptions. It is a world of perceptions, albeit simple ones. The various objects upon which the vision of the child may be casually and very temporarily fixed are seen in their settings. The world the child looks upon is a conglomerate, and because everything that is seen is concrete and separate and yet all of the things that are seen are related to each other because there is nothing to separate them, a world in which everything is related and everything is separate but the connections or lack of connections are not based upon any principle of association or dissociation, in which synthesis and relationship is absent, the qualification syncretistic may be used and the child therefore be said to look out upon a world at first which is a syncretistic conglomerate and which like the word *ma-ma* has to be subsequently analyzed into its separate parts, and then in parallel fashion when the analysis has been completed the various parts of the analysis, such as words of a sentence or letters of a word, are then freed for combinations in new relations, so that, as I see it, the fundamental law here is that analysis and synthesis go hand in hand from this original starting point in the syncretistic conglomerate.

It is generally recognized that the change that takes place in thinking and which is reflected in language is a change from the concrete to the abstract, but this is a half truth. Storch gives an example from the language of savages. For them three boats of one tribe and two boats of another tribe are five boats only under certain circumstances, as for example when the two tribes have undertaken an expedition in concert. Here we find the number five attached to a very concrete situation, and while later on in development five may be attached to any group of five units, whether they be boats, cocoanuts, men, or what not, it would be a mistake to suppose that that represented the only aspect of development, namely, from the concrete to the abstract. At the same moment that the situation of these groups of canoes operating in concert is dismembered, so to speak, so as to release the number five for other uses—at that same moment the whole situation is broken up into a number of component parts which, just like five, can form other associations. Thus the five canoes instead of being

united for a common end may each be associated with an enterprise having a different objective. And so the five and the canoes no longer being necessary to each other are released for wider possibilities of association. Analysis and synthesis go hand in hand. Neither one can occur without the other.

In considering the subject of language it must never be lost sight of that the usual idea that regards it as a vehicle solely for the communication of thought falls far short of the truth. Language has many functions, of which communicating thought is only one, and even with regard to this one function we must never forget that language only imperfectly fulfills it. Every person's own thoughts are peculiarly and exquisitely personal and individual and, too, incommunicable in a real sense. The necessity of living together, social existence, sees to it that there is a certain harmony of action required of the members of the herd. Traffic regulations in a big city are an excellent example of the requirements of a uniform system of signals, perceptions and reactions thereto, but this uniformity need only be statistical uniformity which has an efficiency that is just high enough to prevent the destruction by accident of enough lives to endanger the existence of the group. And so such a degree of uniformity is attained, but this does not insure that everyone who sees the red light sees the same thing, has the same subjective experience which he is able to communicate by the word red. It merely means that roughly speaking a system of signals has been developed which gives a statistically satisfactory result so far as the race is concerned. In fact we can never know what anybody's experience is on seeing red but our own and that depends, as it must in every instance, not only upon the immediate sensation but the total background of the individual's experience, which again the word red when uttered by that individual nucleates and therefore must have a different meaning from the same word uttered by any other individual.

This illustration by way of introduction to the use of examples which might be considered not sufficiently scientifically stated and too superficial, in other words to be near analogies rather than having any closer affiliations, for if my thesis is right we ought to find in the development of language certain phenomena coming regularly to the fore which in the regressive psychoses are re-

animated. A general example of an analogy based upon this hypothesis is that of the child cited by Stern as an example of the volatile character of the child's imagination:

Gunther (3; 2) sitting by my side on the sofa asks me where exactly I wish to go in the train, whistles, hisses and for a couple of minutes really plays at trains. A few moments later, he is sitting on the end of the sofa as on his horse, and now he is either a wild horseman or a cab-driver. He jumps down, pushes two chairs together, gets in, and now he is rocking on a steamer, in a sailing vessel or a motor-boat. He sees the funnel, the smoke, whistles, lands; after a little time, the chairs pushed together are a room "in which he lives," and Hilde comes to visit him, or they are a stable where a number of circus horses are housed. The blind-cord, too, is extremely useful in forming all possible places; he fastens it from the window to the bed, and behind this imaginary partition he then entrenches himself. After a while he is very busy "sewing," i. e., he runs serviette ties through the seat of the cane-bottomed chair.

This example, it will certainly be admitted, if it were of an adult who happened to be a patient in an institution for mental disease, would be put down as a good example of increased psychomotor activity, and if a stenogram were taken of what the little boy said during this period we should have a very good example of flight of ideas. Stern goes on to record what the boy does in the way of drawing, which is repetition of this same movement of attention and interest rapidly from one subject to another accompanied by comments thereon which give this picture of increased psychomotor activity and flight of ideas. He further comments that in children quite the opposite state of affairs maintains from time to time, namely, perseveration, consisting of monotonous repetition, unvarying movements and demands for the same verse of a song over and over again, and the common element which he sees in both of these manifestations is the lack of synthetic power.

Here we have a picture which is illustrative of what I am trying to get at, namely, that we have in the child manifestations of activity and of language which are closely analogous to those manifestations which we recognize as symptomatic of mental disease, in this illustration increased psychomotor activity and probably, although it is not mentioned, a certain degree of euphoria and flight of ideas characteristic of the manic phase of manic-depressive psychosis, and, *per contra*, perseveration, which we are more accustomed to ally with the dementia precox symptomatology.

If my thesis is correct it will be seen that my plea for the study of the structure and function of language after the same manner that one studies a tissue or an organ is well founded, and we have added to the realm of psychiatry a new field for investigation. We have heretofore had our attention solely fixed upon what we conceived to be the thought that the patient was trying to convey by means of language. If we fix our attention upon the language itself we will be able to objectify much which is now pretty nearly wholly subjective. For example, if a patient says he has a pain or other subjective experience he may be telling the truth or he may be malingering. The form in which his statement is cast, however, may be such as to indicate which it is. I was talking only yesterday with a patient recently admitted to the hospital who asked me certain questions. He was particularly interested in who was responsible for his detention and whom he could sue for what he considered to be an illegal restriction of his liberty. We know from a study of children's questions that they do not by any means always represent a desire for information. They have other functions which more particularly can be described as methods of getting what they want, fulfilling their needs. Now this was precisely what this patient was aiming at. It was very obvious after three or four questions and answers that he really was not very much interested in finding out the facts. In the first place, the facts were that he had been properly restrained of his liberty in accordance with the law. He was really looking for an opportunity to find fault. He was anxious to find some concrete individual upon whom he could project his feelings of discontent, his hate. The information I gave him did not assist to this end. He was dissatisfied with it and his comments were exceedingly disparaging. They were to the effect, for example, that he knew just as much after having talked with me as he did before, and as I passed him by I could hear the heavy breathing of an individual who was emotionally considerably disturbed. In this instance we have a paranoid individual seeking through the medium of questions to bring about an opportunity of satisfactory projection, and the form in which the questions are cast and the accompanying emotional expressions and linguistic comments throw this whole thing into a frame of reference which closely allies it to the child's ways and to the child's reasons for asking questions. When the

child is forbidden to do something and he asks the question why he cannot, it is not so much for the purpose of finding out the reason as it is for the purpose of more or less confounding his adult persecutor and perhaps thereby gaining his desires.

It should be possible, if my reasoning is correct, to formulate more closely the analogies between child thought and language and psychotic thought and language, to cast them both in the same mold in accordance with the same principles. While the study of this subject is still comparatively young and the whole field has not been brought under accepted formulations, still there is already enough material which is more than suggestive that what cannot be done today can be supplemented in the future. For example, the study of child thought and language indicates quite clearly to Piaget a number of characteristics which I am sure almost any psychiatrist will admit are at least highly suggestive of similar characteristics among the psychotic, for example, the child's words are much closer to action than the adult's. Our theory of the psyche is that it is built up in that period of delay between perception and action, and if this is true we should expect words of a child, in whom the psyche is still rudimentary, and the words of the psychotic, in whom the inhibitions of the psyche have been removed, both to express themselves more closely in connection with action. This, of course, we know to be a fact, particularly in manic types of reaction, and it is a part of the tradition of the hospital that patients speak out their thoughts. They tell us, in other words, what they think about us really much more spontaneously than do our associates outside. Another characteristic of child thoughts is that they are "impervious to experience." They are purely personal. The child needs no verification. This again is the sort of thing we find in the deluded patient. We are unable to change the delusions by argument. The subjective certainty of the patient is sufficient for him. He needs no verification and he requires no proof from our point of view. It is for this reason largely that the first talk of the child is cast in the form of a monologue, because he is sufficient unto himself. He is not in need of the cooperation or the interest of other people, and if he thinks of it at all he assumes that they think the same way he does. We are at once reminded of the inaccessibility of our patients, who go on talking about their own ideas, elaborating their

delusional system, very much in the monologue form, and with whom it is almost impossible to form any contacts. This is the characteristic difficulty met with in the so-called narcissistic psychosis. It is the egocentricity of the patient, which is on all fours in importance with the egocentricity of the child, except that the child in the natural course of development is finally jostled out of his egocentric attitude of mind. His world is constantly being invaded by others, and his language gradually takes on the characteristics which are necessary in order that he should exercise some control over this environment by placating, bullying, convincing or otherwise influencing the persons about him. All of these characteristics convince Piaget that the thought of the child is intermediate between autism and socialized thought; and while the process of socialization is a long one and takes many years, it is frequently rapidly destroyed in the psychoses and we find the psychotic patients regressed to the period of autistic thought and egocentricity, sufficient unto themselves, inaccessible to those about them.

This egocentricity has other characteristics in the child which are reflected in the psychoses. A predicative judgment such as "Paul is a boy" is typical of such an egocentric state of mind. It is not only egocentric but in its form it is absolutistic. It does not brook contradiction, nor is it disturbed by contradiction. Such a judgment of relation, however, as "Paul is my brother" presupposes at least two points of view, Paul's and my own, and requires a wider field of attention. It is the beginning of a thought process which results in logical thinking, thinking in terms of relation and thinking which ultimately will permit the subject to objectify his own ideas and thought processes and examine them. It will be seen that this absolutistic way of thinking, this way of thinking that is insensible of contradiction, this way of thinking which is expressed by these egocentric forms, is characteristic of the delusion. The delusion cannot be contradicted. It is stated as a fact. It is incapable of objective examination by the subject. Rules of logic cannot be applied to it. The patient is inaccessible.

We have in this state of affairs a type of thinking which has been called by various names. Lévy-Bruhl speaks of pre-logical, Piaget speaks of pre-causal, and because reason and logic do not enter into it but it is dominated by feeling and desire it has been

called, in opposition to rational thinking, irrational thinking. In the child it is due to the inability to handle the logic of relations, the narrowness of the field of attention, synthetic incapacity and juxta-position of ideas rather than their synthesis. It is a causality which in this sense is psychological rather than logical or dependent upon the realities. In such situations we do not find in the child language such words as "because" and "therefore" but the relation is indicated by "and" or "and then." This is an indication that we are dealing with the phenomenon of syncretism, which involves the expression of the perpetual assimilation of everything to subjective schemes. The egocentric tendency replaces adaptation to the external world by assimilation to the self. Syncretism results in what has been described as "immediate analogy," the prompt, unhesitating identification of new objects with old schemes. "Why does the sun not fall down? Because it is hot." When a pebble is put in a glass of water the level of the water rises. Why? Because the pebble is heavy. If wood is used the water rises because the wood is light. Syncretism therefore produces: immediate fusion of heterogeneous elements, an unquestioning belief in the objective intrainplication of elements condensed in this way. It is thus accompanied by a tendency to justify things at any price. The child can always find a reason. The idea of chance is absent from the mind of the child, and this is one of the principal reasons for the phenomenon of precausality. It would seem unnecessary to call attention to the similarity, at least, of this state of affairs with the way of thinking and expression of the delusional psychotic. This concept of precausality helps us to understand the form in which the thought is cast in those patients who have regressed to the point where the distinction between their own ego and the outside world is very largely wiped out, at least in certain areas of their thinking. It is again the way of thinking of the child, whose explanations are neither logical nor spatial and who conceives the world as an assemblage of willed and well regulated actions. The child, as it were, projects explanations upon Nature in accordance with a sort of internal model so that everything can be explained psychologically. There is no differentiation between the self and objective reality, and the animistic ideas of the precox are expressions of this way of thinking.

There is one other analogy which seems to me very significant, and that is that the child lives in two worlds. As its thinking is egocentric and autistic it is in accordance with the law of the pleasure principle and knows of no adaptation to reality, but rather deforms reality to its own purposes. Reality thus is infinitely plastic for autistic thinking, for the child is ignorant of that reality which is shared by all and which therefore destroys illusion and enforces verification.

The two worlds that the child lives in are the world of play and this world of reality which is beginning to insist upon recognition, and it passes swiftly and easily from one to the other, from a state of belief to a state of invention or play, so that these two worlds appear to a child as equally true. The world of egocentric thinking and the world of socialized thinking do not seriously interfere with one another, and this bi-polar state of the child's mind is possible because there is no demand for inner unity for it is only in relation to others that we are obliged to unify our beliefs. This surely is the child analogy for the splitting of consciousness which has become such an important criterion in schizophrenia, and in accordance with the child psychologist Piaget it belongs in the period from 7 to 8 years of age.

If my thesis which I have attempted to illustrate thus far is correct, namely, that language, using the term broadly to include gestures, emotional expressions, drawing, develops in accordance with certain laws and that the regressive psychoses stand to reanimate the various stages in this progressive evolution, then it would be exceedingly interesting and important to discover whether any of the organic disorders present like analogies which make one think that we have again in the realm of structural defect a progressive slicing off, as it were, of the more recent and higher accomplishments and the releasing of the simpler and earlier ones. It seems to me that in that enormously complicated group of symptoms to which we give the generic term "aphasia" may be found many such illustrations, and while I do not pretend to understand how the organic destruction of an aphasia, dependent as it is upon serious massive injury, the result often of large areas of softening in the drainage territories of cerebral vessels which have been occluded by a thrombus, can bring about this regular regressive symptomatology, yet there are indications that somehow

or other it does. As one of the most general examples I might cite Marie's Test, which is to give a patient a series of things to do consecutively, such as to walk across the room, pick up a book on the table, turn it over and put it back, walk around the chair and come back again—such tests as that, which were calculated to show deterioration if there were only two or three or four orders and they were simple ones and could not be carried out. Let me quote from Stern the following and see how close is the analogy:

When the child at eighteen months begins to take little messages from its mother to the maid, it requires not only to remember the words but also the fact of its errand all the way to the kitchen. Even this little distance offers so many distractions, and the child's imagination may meantime have followed so many side issues, that we must not wonder if the determining thought, "I have to take a message," has meantime lost its power.

This is certainly a striking analogy, but it is essential to quote further in order to see how really close it is:

But it is not only fresh and outer impressions that may destroy the dominant interest; hindrances in the performance of a given object may arise from within as well.

For it is only in very easy tasks that the will is able to be fixed upon their fulfillment. If more difficult demands are made, it must first fix and attain intermediate aims, *i. e.*, several dominant interests of a lower order must be pressed into service, without, however, losing sight of the chief interest. Almost all serious forms of activity are in adult life composed of such mingled, diverse, superior and subordinate means and aims, whilst in childhood such systematic arrangement is but weak.

Again and again, we may notice in the child the most striking disparity in intention and execution, and this, indeed, not because the intention was lacking in earnestness, but because the first aims used to the last thread all the energy available in the dominant interest. The child comes to a stop at some intermediate aim, turns it into the main object, and connects with it others quite different not belonging to it at first; in short, his action is a striking instance of the principle of the "heterogeneous character of aims" as propounded by Wundt. In addition to the difficulty of the task, its duration may prove a stumbling-block for the dominant interest.

It seems that here we have without doubt a very close analogy between the disintegration of the adult mind which had previous to the cerebral insult attained a certain power of continuity of action and concentration of thought, and the way in which the child reacts who has not yet attained to this degree of development. Let me give a few other simple examples. In syntactical aphasia

the comprehension of the meaning of words is always in excess of the ability to use them, which is quite in harmony with the development of the comprehension of words by children. We find, too, in this form of aphasia a speech so badly disordered that the patient reproduces only a jargon, which reminds us of the still earlier phase in the infant of babbling. Then we find in nominal aphasia definition by use, which is so familiar to us in the Binet tests. In this type of aphasia there is a loss of power to use names, so that if the patient is asked, for example, what a pair of scissors is, being unable to evoke the word "scissors," he will reply, "What you cut with." Drawing from memory in this and also in semantic aphasia is practically impossible, a condition, too, which we find in early childhood. It may not be quite fair to compare the two, but the net results are strikingly alike. With the child the defect is dependent very largely upon the fact that he reads into reality his own phantasies and therefore when he is asked to draw from memory something that he has observed he does not do it well or at all because he really has not observed reality. He only used reality as a background for his phantasies. What seems to me quite a striking illustration is that of the aphasic patient who is unable to repeat movements of one sitting opposite him, but when the same person sits behind him and he can see from reflection in a mirror the repetitions are well performed. This is on all fours with the difficulties that children have in learning the significance of right and left. For the child at first right and left are entirely subjective experiences and are considered only from his point of view. It is several years before he is able to consider them from the point of view of another person, and quite late in his stage of development before they are considered from the point of view of things in themselves. These constitute three stages in the appreciation of right and left in accordance with Piaget, and correspond to ages 5-8, 8-11 and 11-12, respectively. Right and left, like all other adult abstract concepts, are originally concrete and are referred to specific objects, so it takes some time for this concrete stage to be replaced by the abstract stage in which right and left may be applied to anything and is an experience of relation so that a given object may be on the right side of one thing and on the left side of something else. The aphasic patient mentioned evidently had regressed to a period where he had difficulty in dealing with these concepts at the ordinary adult level.

The drawing of children is very instructive. I have already mentioned synthetic incapacity, and this is shown particularly well in drawing. When objects have been broken up and synthetic incapacity renders their synthesis impossible, what gathers the juxtaposed elements into a group? It has been suggested that it is a relation of membership and not of inclusion, and so an arm drawn alongside a mannikin is conceived by the child as "going with" the mannikin and not as "forming part of" its body. So a child will draw a bicycle. The wheels, the pedals, the seat, the handle-bars will all be drawn, but they will not be drawn in their proper relations. They will not be joined one to another at all but they will be drawn as separate parts, perhaps overlapping, perhaps not. There is a lack of synthesis. This is what we see in the aphasic. The drawing as well as the speech tends to become much more concrete and less abstract. This regression is in the same direction in which the language has developed in the individual and also in which it has developed in the race, for the languages of primitive peoples, "always express their ideas of things and actions in the precise fashion in which these are presented to the eye or ear."

These last examples from the realm of the organic have an added significance to that already given them. They not only indicate that the organic regression follows the reverse of the law of development but we see at once we have made this statement, that it is the same statement that we made with regard to the law of regression of function.

This means nothing more nor less than that the law of dissolution or devolution or dedifferentiation, whichever you will, is the same whether it operates in the realm of the functional or of the organic. This seems to me a conclusion of great significance. The organicists and the functionalists have been occupying two camps, separate and distinct from one another. They have represented the ambivalent opposites of the possibilities of interpretation of pathological phenomena. I have always believed that this condition of affairs was in reality an artifact, that it represented a pseudo-problem, namely, the distinction between the functional and the organic. I have never conceived that these two aspects could be separated, and I believe that one of the very important things that will happen in our field of inquiry will be to bring these two

groups together on a common basis so that they will each realize the significance of their own point of view in reference to the other point of view, and the fact that either point of view is only one aspect or one way of looking at the situation. When it is realized that this law of regression cannot only be applied in these two spheres but its application is possible to situations where there is localizable injury, we can see how various problems will receive illumination. There is the whole field of schizophrenia, for example, which seems to be in the borderland between the functional and the organic. There is the field of sleep disturbances, which seem to be represented on the one hand by entirely functional phenomena and on the other hand by phenomena that we must consider as organic, such as those seen in encephalitis. If we can hitch up these ways of looking at phenomena our prospect of appreciating and understanding at least this material which stands midway between the two will be greatly increased.

The conclusions to which I would invite your attention as being of the most moment are as follows:

First, The mind does not develop by an additive process. The world that the child looks out upon from the very beginning is a syncretistic conglomerate;

Second, The development from this original state is by the way of a pair of ambivalent opposites, analysis and synthesis, which develop concurrently and along parallel lines;

Third, The law of regression is the same for both functional and organic states; and

Fourth, Functional and organic are merely different ways of looking at phenomena.

BIBLIOGRAPHY.

- Head, Henry: Aphasia and Kindred Disorders of Speech. Vols. I and II. New York, The Macmillan Co., 1926.
- Jespersen, Otto: Language, Its Nature Development and Origin. New York, Henry Holt & Co., 1924.
- Lévy-Bruhl, Lucien: How Natives Think. New York, Alfred A. Knopf.
- Piaget, Jean: The Language and Thought of the Child. New York, Harcourt, Brace & Co., 1926.
- Judgment and Reasoning in the Child. New York, Harcourt, Brace & Co., 1928.
- Stern, William: Psychology of Early Childhood. New York, Henry Holt & Co., 1924.

- Storch, Alfred: *The Primitive Archaic Forms of Inner Experience and Thought in Schizophrenia.*, Nerv. & Ment. Dis., Monograph Series No. 36. Washington, Nerv. & Ment. Dis. Pub. Co., 1924.
- White, William A.: *An Introduction to the Study of the Mind.*, Nerv. & Ment. Dis., Monograph Series No. 38. Washington, Nerv. & Ment. Dis. Pub. Co., 1924.
- The Meaning of Disease*, Chapter X, Baltimore, The Williams & Wilkins Co., 1926.
- The Language of Schizophrenia.* Archives of Neurology & Psychiatry. Vol. 16, pp. 395-413, October, 1926.

DISCUSSION.

DR. GEORGE S. SPRAGUE (White Plains, N. Y.).—Dr. White has brought before us a suggestion that is of profound interest and significance in the study of psychiatry and especially I think, in relation to an understanding of some of the problems of thought disturbance in the psychoses, particularly schizophrenia. Perhaps we do not know Arabic, but do we not understand the Arab who is angry at us, or who is intriguing? The whole field of gesturing has to be included in the topic of language. Language is not an individual affair; it is rather something that betokens an understanding, a relationship between two people; not "I talk," but "I talk to somebody," whether it is done with the voice or the muscles.

It has always seemed to me that there are elements in the psychotic's expression of thought which deserve a careful correlation with the different methods of thought expression in different languages. What do we mean by "expression of thought?" Is it intonation? Consider, then, the Chinese in which the merest change of intonation, of inflection of a word, changes the whole meaning of the word. The methods that we are accustomed to use for expressing incredulity or emphasis have, in Chinese, an actual grammatical significance. Is it a question of general attitude; as of politeness, or of derision? In that connection it is interesting to consider the entirely different spoken languages of the Japanese, who use different pronunciations for the same ideographs according to whether their meaning is one of politeness or otherwise.

There are a great many such comparisons that it might be helpful to know in understanding the disturbances of speech of the schizophrenic. The variations of word order for instance compare possibly, with the method of thought expression of the Chinese language, in which the change of word order alters entirely the properties, or even actually the parts of speech, of the individual sound.

The question of language being a growth, a development with increasing grades of complexity is, of course, familiar to us all, we need only to point out that a person thinks naturally in the most general concepts that he is able to employ. When we, used to such sights, see a thrashing machine, we don't see immediately the separate parts, the wheels, the rods, the belts; we see the whole picture in so far as we know it, the "greatest common

divisor," shall we say, of the total mass of concepts; whereas, to the child who has never comprehended so complex a total concept, the smaller concepts represent his level of thought

I hope that we are going to have in the near future studies of this sort which will perhaps correlate the differences of disturbance of schizophrenic thinking with different methods of thought expression, in various languages.

DR. A. A. BRILL (New York, N. Y.).—I hesitated to participate in the discussion because Dr. White's very interesting paper is so very comprehensive that one does not know just where to start and what part to discuss. I shall, however, confine myself to just one element of Dr. White's paper, namely, the child method of expressing language and its resemblance to the schizophrenic methods of expression. Those of us who have studied the problem fully agree with Dr. White. We see this mode of expression even in those schizophrenics who maintain themselves at large. For the last few years we have been deluged with a mass of literature; I should like to refer you to a very interesting work called "Tender Buttons," by a woman who is well known internationally for her peculiar productions. She has written a number of works which, if you read them, you will find resemble very much the schizophrenic flow of thoughts. As a matter of fact, you would not be able to tell the difference between her works and schizoid-manic productions. I have taken pages from her various works and put them parallel to the productions of schizoid-manic patients. I showed them to psychiatrists and to lay people and nobody could tell the difference between them. There are quite a number of writers who are following in her footsteps, and one can find many good examples in the poetic productions of *vers libre*. Most of those poets belong to the schizophrenic type who can barely maintain themselves.

Occasionally one also encounters this mode of expression in great minds. For instance, Abraham Lincoln had a habit of playing with words. The moment he heard the word "facsimile," he would at once exclaim "sic family."

I am now working on a paper on the origin of poetry, strange as it may seem to you. I was instigated to do it by a number of my patients who told me that at certain times they feel a strange impulsion to poetize in an infantile way. They just play with words and sounds. As you know, children, when they are beginning to talk, do not use words as words, but as sounds, and, as was mentioned by the previous speaker, we see similar phenomena in various languages. Thus the same words differently modulated may express contrasts, and traces of this may even be found in modern tongues. Some of my patients have never given up the original playing with sounds or clanc; they frequently use the same consonants, and sometimes compulsively repeat meaningless words in a sort of rhythm. These patients when they become psychotic, particularly the schizoid-manic types, invariably resort to these expressions. Most of them are ashamed of these productions and only tell about them reluctantly. These nonsensical ex-

pressions can usually be analyzed and traced to some infantile playing with words or to some expressions that happened to appeal to them on account of some sound or some affective association, pleasant or unpleasant. I feel highly indebted to Dr. White for showing us the intimate relation between the normal expressions and the linguistic disturbances in the organic and psychotic languages.

PRESIDENT ORTON.—I would like to add just a word of interest to Dr. White's offering from the standpoint of my own current interests which are those in the difficulties of acquisition of written language particularly, but also of verbal language in children. One can parallel almost everything Dr. White has said to us in the patterns of the special disabilities. It is sometimes startling to see how clear-cut the parallelism is there and also we begin to get some beautiful illustrations of what the patterns are that determine some of these infantile implants which may later come back. Some of them are quite superficial and obvious; some of them, frequently many of them, are determined by what I believe to be a lack of establishment of an unilateral cerebral dominance, and we see the evidence of this in the reversal of direction in the reading so the child reads backwards through the words or twists a pair of letters in the word or twists one of two syllables within the word or reads one in one direction and one in the other. Those things are obvious, demonstrable things which one can, by merely listening to the child's errors or analyzing his spelling, make out with conciseness. That particular factor is a fairly prominent one. It is not a rare individual matter but can be demonstrated to be present in approximately 2 percent of all school children who are trained by the older methods of reading and in approximately 6 per cent of those school children who are trained by the modern method which Dr. White has suggested to us as much nearer the normal. It may be nearer the normal for the average child, but for this particular group of cases it forms an obstacle to acquisition which is very striking.

Moreover, in many of these children who have never acquired an adequate association of the written word with its auditory counterpart (have not linked them together), you can find just as good a jargon aphasia, just as good a group of neographism or neologisms, as you can see in cases of schizophrenia. I think the parallelism there is exceedingly interesting. Moreover, you will find curious individual implants. For example, one boy in attempting to read for me the word "hostility," produced the word "hostlostily." In order to find out how he got "hostlostily" from it, I asked the mechanism. He had been taught the modern method. He had been taught the word "lost" as a word, but not "host." In thinking of "hostility," he had to first take "lost" and take the "l" off and put an "h" in front. He got "host." But the "lost" was still reverberating in his mind and when he came to the following "l" in "hostility," the "lost" recurred, and he got "hostlostily." That reverberation in the mind apparently is quite a common source of spelling and reading errors.

Again, you will find whole word implants, where a child has not been taught the alphabet, cropping up, or you may find a word that is entirely out of harmony with the word he is attempting to spell. A small girl spelled for me uncle, w-a-l-c-e-l. The c-e-l origin is obvious, but the w-a-l I was not able to trace back until in talking with her mother I found she had an Uncle Walter who was familiarly known in the family as Uncle Walt. Here the association implanted in her spelling w-a-l first and c-e-l for uncle afterwards.

Is there any further discussion? If not, I will ask Dr. White to reply.

DR. WILLIAM A. WHITE.—I have nothing further to say.

THE EMPIRICAL DETERMINATION OF CERTAIN SYNDROMES UNDERLYING PRÆCOX AND MANIC-DEPRESSIVE PSYCHOSES.*

By THOMAS VERNER MOORE, PH. D., M. D.,

Professor of Psychology, Catholic University of America; Director of the Clinic for Mental and Nervous Diseases, Providence Hospital, Washington, D. C.

If a clear and definite pathology, such as we have for valvular diseases of the heart, were established for the various mental disorders, such a study as the present would be superfluous. But we have as yet only the vaguest notions about the pathology of the mind, and as a result the diagnostic entities of psychiatry are so poorly defined that the attempts to associate physical symptoms with definite mental conditions have been most disappointing. Likewise, psychological measurements of memory, association, reaction-time, etc., have been difficult of interpretation, when they had to be attached to the vague, overlapping, indeterminate entities of clinical psychiatry.

The present study is an attempt to see whether or not it is possible to determine in a purely empirical manner any syndromes at all in the mental disorders. The final results only are here reported. But the preliminary stages were:

(1) The working out of a schema for measuring quantitatively various emotional manifestations of the mental disorders. This will be published shortly in our *Studies in Psychology and Psychiatry* (Williams & Wilkins, Baltimore, Md.).

(2) The development of a method for measuring reasoning, perception, the span of memory and the rate of forgetting as indicators of cognitive defect. The reasoning test was published recently.¹ The tests for perception and memory were used on a previous occasion.² We are well aware that the tests we have used are capable of being vastly improved, but they have served their present purpose and clearly defined certain psychiatric syndromes.

* Read at the International Congress of Psychology, Yale, September 5, 1929.

They may, therefore, be employed for the detection of other syndromes in the future; and that too with considerable reliability.

(3) The recording of a number of symptoms as present or absent. A quantitative measurement of these was not attempted, partly because of intrinsic difficulty, partly because the labor of the research was necessarily limited.*

In all, 367 patients were examined though not all had every item recorded. The items most often lacking were the cognitive measurements. This was due mainly to the time taken by such tests, partly to the necessity of full cooperation on the part of the patients. Refusals were surprisingly seldom, for after a little an interview with the psychologist came to be regarded as an opportunity of demonstrating the soundness of one's mentality. About 208 patients were given the cognitive tests and about 350 had the full emotional record.

After the measurements had been completed the attempt was made to intercorrelate 41 symptoms finally selected as a basis for the empirical determination of the syndromes of psychiatry.

When curves of distribution were made it was found that in all the emotional symptoms we had the positive side only of a normal curve with the negative cases heaped up at the zero point. This was due to the assumption made in our quantitative schema that, *e. g.*, the depressions would show a mean frequency at a point indicating a depression of average intensity and taper off on either side to zero depressions and those of maximal intensity. Depressions, however, do not do this, nor do any other emotional symptoms in our schema. The maximum frequency is not far from our zero point. This is in itself a very interesting fact and indicates that normal mental life is at a zero point of emotional equilibrium.¹ When a psychotic condition develops, the plane of emotional equi-

* I must here make acknowledgment to the authorities of Mt. Hope Retreat, Baltimore, Md., for the appointment of a psychologist, Miss Gertrude Reimann, to carry out the tests and measurements as a part of the routine examination of patients; to Dr. William A. White, who allowed me to examine a number of patients at St. Elizabeths Hospital; to the rector of the Catholic University for a grant of \$500; and to Sister Rosa McDonough for considerable help in the calculations; and to Miss Evangeline Sheibley. The sorting of the cards for the correlations, which would have been an almost impossible task by hand, was kindly done for me by the Remington Rand Business Service on the Powers Sorting Machine.

librium is tilted to one side or the other. Slight tilts are more frequent than any other just as small excesses of the probable number are more frequent in the tossing of coins. An abnormal emotional condition is, therefore, not a new psychological growth but an excess or defect of a normal mental condition.

It might have been possible after some study to determine the precise opposite of depression, "shut-in," irritability, euphoria, etc., but unless this were done without any ambiguity the results would have been seriously clouded. Consequently, we made use of the data obtained by the quantitative schema we had developed. It enabled us to clearly define the symptoms studied and to say whether or not a patient manifested one of these symptoms in definite excess. This information led to the formation of fourfold tables in which patients were placed with a fair degree of certainty in one of the four compartments that compared the presence and absence of two traits. We were thus enabled to make use of Pearson's method of tetrachoric correlation. The correlations so obtained are theoretically the same as the product moment r when the distribution is normal.

There are 820 possible intercorrelations for a total of 41 symptoms. Naturally we are interested mainly in those of significant magnitude. To select these without having to go through the labor of calculating r the following expedient was used.

Consider for a moment the accompanying fourfold table for "Auditory Hallucinations" and "Loss of Finer Sensibilities."

	Auditory Hallucinations		
	Absent.	Present.	
Loss of Finer Sensibilities, <i>Absent</i>	160	138	298
Loss of Finer Sensibilities, <i>Present</i>	13	34	47
	<hr/> 173	<hr/> 172	<hr/> 345

In the group of insane patients that had no "loss of finer sensibilities" $\frac{138}{298}$ showed auditory hallucinations. Taking this as the probability of the occurrence of auditory hallucinations as a chance phenomenon in the insane we would expect, if the same chance obtained in patients with "loss of finer sensibilities," an incidence of 22.224 in our group of 47 cases in the above table. (Incidence =

probability times number of throws plus one.) As a matter of fact we find 34 cases. Would this excess of 11.776 be likely to occur by chance? The probability of this excess as a chance phenomenon is obtained by calculating its sigma value.

$$\sigma = \sqrt{p(1-p)n}.$$

That is to say σ equals the square root of the probability that the event will occur (p) times the probability that it will not occur ($1-p$) times the number of cases or throws.

In the case $\sigma = \sqrt{(.46) (.54) (47)} = 3.417$.⁴

Various tables⁵ are available for the calculation of the probability of an error or excess which is given in terms of σ . The probability of such an excess as a mere chance event is only about three in ten thousand. It is, therefore, likely that any correlation that may be found between the above two symptoms is not due to chance. As a matter of fact the tetrachoric r amounts to .352.* By means of this method we spared ourselves the labor of calculating the tetrachoric r wherever the excess of the incidence divided by the

sigma value $\left(\frac{x}{\sigma}\right)$ was not above 2.67 that is above a chance incidence of about one in a hundred.† Some few with smaller values were later calculated in determining the tetrad differences discussed below.

The table of probability values and correlations obtained in this way is very suggestive of many interesting problems but it does not of itself enable us to pick out the clinical entities of psychiatry. We first thought of taking each symptom in turn and paralleling its associates with those of other symptoms, hoping in that way to pick out symptoms that group together or are mutually exclusive. Or one might arbitrarily choose a symptom that figures in classic

* In calculating our tetrachoric r values we neglected terms in the formula involving powers above r^2 ; for unless the correlation is very high these terms only subtract a few units in the third or fourth decimal place. Such quantities are negligible for our purposes.

† In any fourfold table there are always two ways of calculating $\frac{x}{\sigma}$. We always chose the one which involved the greater number of cases for the fundamental p value.

discussions of the psychoses and take its associates as the determinants of the condition in question, *e. g.*, depression.

It was thought, however, best to apply the tetrad difference criterion of Spearman to the knotty problem of the mental disorders.*

According to this criterion, if a, b, p, q , represent four abilities or variable quantities of any kind, and r stands for the correlation value, then there is one common underlying factor in all four specific manifestations if $r_{ap} \cdot r_{bq} - r_{aq} \cdot r_{bp} = 0$.

Furthermore, all possible combinations of these correlations in any table of four or more variables give zero values within the limits of sampling error.

In the application of such a formula to a series of 41 variables it is out of the question to calculate the tetrads for the whole table which would mean the solution of hundreds of thousands of equations. There is, furthermore, little likelihood that there is one and the same general factor underlying all the psychoses. The practical method of procedure is to take each symptom and look for its highest correlation and then its next highest. Having found a series of four such closely related symptoms, one arranges their table of intercorrelations. If such a table manifests an evident "hierarchy" it is worth while calculating the tetrads. By hierarchy Spearman means the proportionate gradation of all the columns and rows from higher to lower values.* If the tetrad values fall within the limits of sampling † one then seeks a fifth variable and so on. In our material we were unable to group together more than five variables.

In this way we have been able to pick out eight general factors that are present in the psychotic conditions from which our patients suffered. In all probability they are fundamental psycho-biological

* Consult the tables given below and it will be seen that with few exceptions all the values grade down from the upper left-hand corner below and to the right. All columns from above downward and all rows from left to right grade more or less proportionately downward. This hierarchy of values was Spearman's original criterion for the presence of a common factor. When, however, the hierarchy was imperfect there was no way by the old technique of telling how much could be allowed to chance errors of observation.

† According to Spearman no tetrad difference should be five times its probable error. This is a reasonable criterion when there are a large number of variables, but is rather lax for only four.

conditions that are involved in the "manic-depressive" and "præcox" psychoses. Having found these conditions or general factors we wished to go further and discover a way for measuring the degree in which any given patient manifests them.

The first step towards this end was given by Dodd[†] though he merely conceived of it as a means of demonstrating the presence of a general factor.

The second step was taken by Sister Rosa McDonough^{*} who recently applied the technique to the measurement of character traits.

Dodd's work was founded on the method given by Spearman for measuring the correlation between a specific ability and the underlying general factor.* Having determined these values he used the ordinary formula for partial correlation and obtained the correlation between the specific abilities after the general factor had been partialled out. If a general factor is present and the specific factors do not overlap (that is, are truly independent) then the average of all these partial correlations will approximate zero.

In the paper above referred to, Sister Rosa McDonough proceeded from this point and (1) conceived of the general factor as the criterion in a multiple regression equation; (2) determined the appropriate weights for the individual measurements so that the sum of the weighted values would be the measure of the individual's participation in the general factor.

We have done the same thing for the psychotic symptoms that are indicators of underlying general factors in the tables that are herewith presented.

Unfortunately, however, for a number of our symptoms we have as yet no quantitative measure[†] and our results in these instances only show how accuracy may be obtained by a psychological study of the symptoms of the psychoses. Symptoms marked with an asterisk in the following tables of intercorrelations had no quantitative measure. The limit of the probable error is taken, for convenience as that of the product moment formula. The tetrachoric probable error is about double that of the product moment.

* Cf. *The Abilities of Man*, Appendix, p. xvi.

† Furthermore, except in the cognitive symptoms all negative values are recorded as zero. Practically, however, this need not be of serious difficulty for it would be possible to measure quantitatively the symptoms actually presented by any individual.

TABLE I.

THE COGNITIVE GROUP.

A. INTERCORRELATIONS (TETRACHORIC).

	Reasoning.	Perception.	Total memory.	Memory ratio.	Shut-in.
Reasoning593	.515	.358	.398
Perception593380	.338	.344
Total memory515	.380339	.207
Memory ratio358	.338	.339189
Shut-in398	.344	.207	.189	...

B. TETRADES FOR INTERCORRELATIONS IN TABLE I.

Average of tetrads.....	.034
P. E. of tetrads.....	.021

C. CORRELATIONS OF VARIABLES WITH G AND CORRELATIONS WITH G
PARTIALLED OUT.

<i>r</i> _{ag}853	.720	.583	.481	.425
------------------------------	------	------	------	------	------

Partials.

Reasoning058	.042	.114	.075
Perception058071	.014	.060
Total memory042	.071082	.055
Memory ratio114	.014	.082019
Shut-in075	.060	.055	.019	...

Average of all the partials.....	.059
Limit of the P. E.....	.046
Significance ratio	1.274

D. β VALUES AND MULTIPLE CORRELATION COEFFICIENT. β values

Reasoning55	Multiple correlation coefficient.....	.914
Perception28		
Total memory14		
Memory ratio12		
Shut-in06		

Regression equation

$$\frac{G\text{-factor}}{.00} = (.55) \frac{R - 41}{22.8} + (.28) \frac{P - 38}{14.9} + (.14) \frac{T. M. - 44}{17} \\ + (.12) \frac{M. R. - 32}{25.1} + (.06) \frac{S. I. - (-2.9)}{29.8}$$

TABLE 2.

THE CATATONIC GROUP.

A. INTERCORRELATIONS (TETRACHORIC).

	Mutism.	Negativism.	Refusal of food.	Stereotypism of attitudes.
*Mutism817
*Negativism817687	.627
*Refusal of food.....	.687	.666	.666	.628
*Stereotypism of attitudes.....	.627	.628	.572	.572

B. TETRADES FOR INTERCORRELATIONS IN TABLE 2.

[†] 1234035
[†] 1243062
[†] 1342027
P. E. of tetrads.....	.016

C. CORRELATIONS OF VARIABLES WITH G AND CORRELATIONS WITH G
PARTIALLED OUT.

<i>r_{ag}</i>896	.883	.773	.717
	Partials.			
Mutism124	.020	.050
Negativism124056	.016
Refusal of food.....	.020	.056040
Stereotypism of attitudes.....	.050	.016	.040	...
Average of all the partials.....				.051
Limit of the P. E.....				.037
Significance ratio				1.397

D. β VALUES AND MULTIPLE CORRELATION COEFFICIENT. β values

Mutism44	
Negativism27	
Refusal of food.....	.20	
Stereotypism of attitudes..	.16	
		Multiple correlation coefficient... .952

TABLE 3.
THE UNINHIBITED OR KINETIC GROUP.
A. INTERCORRELATIONS (TETRACHORIC).

	Stereotypism of actions.	Destructive.	Giggling.	Talking to voices.
*Stereotypism of actions.....667	.523	.566
Destructive667402	.395
Giggling523	.402366
Talking to voices.....	.566	.395	.366	...

B. TETRADES FOR INTERCORRELATIONS IN TABLE 3.

^t 1234038
^t 1243017
^t 1342021
P. E. of tetrads.....	.019

C. CORRELATIONS OF VARIABLES WITH G AND CORRELATIONS WITH G
PARTIALLED OUT.

<i>r</i> _{ag}937	.686	.585	.603
------------------------------	------	------	------	------

Partials.

Stereotypism of actions.....095	.088	...
Destructive095001	...
Giggling088	.001
Talking to voices.....	.002	.032	.020	...

Average of all the partials.....	.040
Limit of the P. E.....	.036
Significance ratio	1.106

D. β VALUES AND MULTIPLE CORRELATION COEFFICIENT. β values

Stereotypism of actions... .78	
Destructive09
Giggling11
Talking to voices..... .08	
Multiple correlation coefficient.... .949	

TABLE 4.
MANIC GROUP.
A. INTERCORRELATIONS (TETRACHORIC).

	Irritable.	Tantrums.	Destructive.	Euphoria.
Irritable611	.492	.473
Tantrums611438	.364
Destructive492	.438303
Euphoria473	.364	.303	...

B. TETRADES FOR INTERCORRELATIONS IN TABLE 4.

[†] 1234015
[†] 1243020
[†] 1342035
P. E. of tetrads.....	.018

C. CORRELATIONS OF VARIABLES WITH G AND CORRELATIONS WITH G
PARTIALLED OUT.

<i>r_{ag}</i>865	.716	.586	.524
-----------------------------	------	------	------	------

Partials.

Irritable024	.037	.046
Tantrums024047	.019
Destructive037	.047006
Euphoria046	.019	.006	...

Average of all the partials.....	.030
Limit of the P. E.....	.036
Significance ratio836

D. β VALUES AND MULTIPLE CORRELATION COEFFICIENT.

β values		
Irritable60	
Tantrums23	
Destructive16	
Euphoria10	
	Multiple correlation coefficient....	.913

TABLE 5.

DELUDED-HALLUCINATED GROUP.

A. INTERCORRELATIONS (TETRACHORIC).

	A. H.	B. D.	O. H.	St. W.	D. S.
Auditory hallucinations740	.699	.455	.359	
Bizarre delusions740	.692	.436	.360	
"Other" hallucinations ..	.699	.692	.344	.236	
Stereotypism of words....	.455	.436	.344	.281	
Disorientation in space...	.359	.360	.236	.281	

B. TETRADES FOR CORRELATIONS IN TABLE 5.

Average of tetrads.....	.049
P. E. of tetrads.....	.020

C. CORRELATIONS OF VARIABLES WITH G AND CORRELATIONS WITH G
PARTIALLED OUT.

<i>rag</i>893	.878	.695	.528	.418
Auditory hallucinations204	.242	.043	.035	
Bizarre delusions204	.238	.068	.016	
"Other" hallucinations ..	.242	.238	.038	.083	
Stereotypism of words....	.043	.068	.038	.078	
Disorientation in space...	.035	.016	.083	.078	

Average of all partials.....	.1045 *
Limiting value of P. E.....	.036
Significance ratio	2.90

D. β values

Auditory hallucinations ..	.50	
Bizarre delusions42	
"Other" hallucinations ..	.02	Multiple correlation coefficient.... .952
Stereotypism of words....	.10	
Disorientation in space....	.06	

* Some partials in above table are evidently too high to allow the assumption to hold that there is no bond between the specific other than the general factor.

TABLE 6.
THE CONSTITUTIONAL HEREDITARY DEPRESSION GROUP.
A. INTERCORRELATIONS (TETRACHORIC).

	Anxious.	Depressed.	Tearful.	Previous attacks.	Insane relatives.
Anxious725	.564	.221	.194
Depressed725475	.288	.187
Tearful564	.475175	.104
*Previous attacks221	.288	.175
*Insane relatives194	.187	.104	.127	.127

B. TETRADES FOR INTERCORRELATIONS IN TABLE 5.

Average of tetrads.....	.029
P. E. of tetrads.....	.016

C. CORRELATIONS OF VARIABLES WITH G AND CORRELATIONS WITH G PARTIALLED OUT.

<i>r_{ag}</i>832	.841	.543	.362	.257
-----------------------------	------	------	------	------	------

Partials.

Anxious084	.241	-.155	-.037
Depressed084040	-.033	-.056
Tearful241	.040	...	-.028	-.044
Previous attacks155	.033	-.033038
Insane relatives037	.056	-.044	.038	...

Average of all the partials.....	.076
Limit of the P. E.....	.036
Significance ratio	2.078

D. β VALUES AND MULTIPLE CORRELATION COEFFICIENT. β values

Anxious42	
Depressed45	
Tearful08	
Previous attacks12	
Insane relatives07	
		Multiple correlation coefficient.... .912

TABLE 7.

THE RETARDED-DEPRESSED GROUP.

A. INTERCORRELATIONS (TETRACHORIC).

	Depressed	Retarded	Neurasthenia	Suicidal
Depressed743	.372	.328
Retarded743 *328	.141
Neurasthenia372	.328060
Suicidal328	.141	.060	...

B. TETRADES FOR TABLE 7.

^t 12340079
^t 12430630
^t 13420551

Probable error of tetrads..... .0222 (Spearman's formula)

Probable error of ^t1234..... .0246 (Kelley's formula)

* The correlation between depressed and retarded is so high that when one attempts to work out the partials as in the other tables one gets incompatible results. It is probable that in this group there is one general factor plus a special group factor between depressed and retarded.

THE PROBABLE NATURE OF THE GENERAL FACTORS IN THE PSYCHOSES.

The above tables demonstrate only the existence of certain general factors underlying the specific manifestations of various mental disorders. The bare facts that a hierarchical order comes out in the tables of correlation, that the tetrad differences are zero, within the limits of errors of sampling, that the partial correlations between the specific symptoms approach zero when the general factor is partialled out, while each specific symptom has a significantly high correlation with the general factor, all these facts together may be taken as conclusive evidence that certain general factors exist in the mental disorders.

We are naturally interested in learning something about the nature of these general factors.* We shall deal first and mainly by way of example with the cognitive general factor.

Let us take up in the first place the evident character of the specific symptoms. In the cognitive group with one exception they have to do with cognition. The fact that our statistical technique

* The names given to the various factors were suggested by looking at the general character of the specific symptoms.

has assembled these in one group instead of scattering them indiscriminately among all groups is evidence that a natural law of some kind is responsible for the grouping of the symptoms.

At first sight it seems strange that the symptom "shut-in" should appear with the cognitive group. That it does, must be taken to mean that "shut-in," as a pathological symptom, is an indication of cognitive defect. In our quantitative schema "shut-in" was a measure of the poverty of emotional expression, the mask-like facies so common in *præcox* patients. Now according to the data presented in the above tables "shut-in," as so defined, is an indicator of cognitive defect. Furthermore, it has positive correlations with all the cognitive measures we employed.* It may, therefore, be conceived of as a measure of cognitive defect. But how is this possible?

There is good reason to suppose that intelligence is the fundamental causal factor in emotional experience.⁹ If intelligence is shallow, emotional experience must also be reduced. With a defect of intelligence and of emotional experience there should go hand in hand an impoverishment of emotional expression.¹⁰ If this is the case, it is not surprising that "impoverishment of emotional expression" should appear in the multiple regression equation as an indicator of cognitive defect.

This fact will be of value in understanding the nature of dementia *præcox*. For cognitive defect, as will be seen by Table 8, is related to the *præcox* rather than the manic-depressive psychoses. This table gives us the intercorrelations of the general factors of the psychoses. Examining this we find that cognitive defect has a negative correlation with the retarded depressions and the constitutional hereditary depressions. It has positive correlations with what are probably two phases of *dementia præcox*: the uninhibited and the catatonic. This, along with the fact that "shut-in," the classic symptom of the *præcox*, enters into the syndrome of cognitive defect, is strong evidence of impairment of intelligence in a

* Besides those found in our tables these measures were: (1) ability to detect logical and (2) autistic fallacies and (3) absence of insight. "Detection of fallacies" gives high tetrads when thrown into the cognitive group, probably because this ability involves reasoning and, therefore, is not independent of the other measures, a necessary condition. Absence of insight is such a common symptom in all forms of mental disorder that it is not a good diagnostic criterion.

group of patients that would be ordinarily classified under the heading of *dementia præcox*.

It is not necessary that this impairment of intelligence should be permanent. Thus, for instance, a patient with delusions and

TABLE 8.

INTERCORRELATIONS OF THE GENERAL FACTORS.*

	I	II	III	IV	V	VI	VII	VIII
I. Retarded depression688	-.046	-.119	-.178	-.415	-.441	-.517
II. Constitutional hereditary depression .	.688	...	-.369	-.019	-.439	-.346	-.315	-.117
III. Catatonia ...	-.046	-.369401	.331	.651	.383	.039
IV. Non-euphoric manic	-.119	-.019	.401	...	-.154	.205	.029	X
V. Cognitive defect	-.178	-.439	.331	-.154439	.322	.272
VI. Uninhibited ..	-.415	-.346	.651	.205	.439305	.024
VII. Deluded-Hallucinated ..	-.441	-.315	.383	.029	.322	.305169
VIII. Euphoric manic	-.517	-.117	.039	X	.272	.024	.169	...

* The manic group was split into two; the euphoric and non-euphoric manics. This was suggested because most euphoric patients are irritable but not vice versa; and because the distribution of patients scoring above the mean in euphoria is bi-modal with marked hump at the extreme of the distribution. The retarded depressions are here included but a table giving the multiple regression constants were not calculated because correlation of the syndrome with other symptoms seemed to imply the possibility that the same general factor is involved in the constitutional hereditary depression.

TABLE 9.

COGNITIVE IMPROVEMENT IN "DEMENTIA PRÆCOX" WITH CLEARING OF THE PSYCHOSIS.

	Total memory.	Memory ratio.	Perception.	Reasoning.	Autistic fallacies.	Logical fallacies.	Absence of insight.
October, 192856	.50	.56	.58	.75	.75	+
May, 192938	.075	.28	.14	.50	.38	—

auditory hallucinations, who was "shut-in," talking to voices and manifested stereotypisms of actions and attitudes was diagnosed in October, 1928 (after an acute onset six weeks previous), as suffering from dementia præcox, catatonic form. In the following May the condition had considerably cleared. Table 9, in which the

figures denote percentages of defect, shows a marked cognitive impairment in October, 1928, which had been to a large extent overcome in May, 1929.

If dementia præcox involves an acute or chronic impairment of cognitive function it probably has some kind of organic factor in its etiology, and cannot be regarded as a wholly psychogenic mental disorder.

A discussion of the ultimate nature of the cognitive general factor would lead us too far afield. It is at present being actively investigated by many psychologists.

It might not be out of place, however, to suggest:

(1) The cognitive general factor which is impaired in certain mental disorders is the same general factor that psychologists have found to underlie the most various and widely different psychological performances.

(2) It is likely that this general factor is what has been found to be inherited in various studies of the mental inheritance of genius and feeble-mindedness.

(3) Should the work of Travis and Hunter be confirmed²¹ or any similar correlation between neurological factors and intelligence, it would be possible to specify more definitely what is the nature of this cognitive general factor.

(4) Whatever the nature of this general factor, it cannot be any one of the specific mental abilities which enter into the formation of the tetrads nor their sum total, nor can it overlap to any significant degree with any one of them for this would lead to large tetrad differences which is contrary to the findings.*

* The reason for this statement may be shown as follows. Spearman develops the tetrad equation from Yule's formula for partial correlation (*cf.* Spearman. *The Abilities of Man*, Appendix, p. iii)

$$r_{ap.g} = \frac{r_{ap} - r_{ag} r_{pg}}{\sqrt{1 - r_{ag}^2} \sqrt{1 - r_{pg}^2}}$$

He assumes that when the general factor (*g*) is partialled out the correlation between any two specific factors *a* and *p* will be zero. This reduces the above equation to $r_{ap} = r_{ag} r_{pg}$. By a similar equation between *b* and *q* he comes finally to the tetrad equation $r_{ap} r_{bq} - r_{bp} r_{aq} = 0$. If, now, one supposes that the general factor is any one of the specifics the above equation is meaningless. Assuming that the correlation of a thing with itself is unity the formula for

(5) Many experiments have been made and a great variety of mental abilities have been found to involve an underlying general factor. The general factor is not, therefore, any one of these mental abilities. Furthermore, it is not likely that the general factor in intelligence will turn out to be any known human mental ability.

(6) That which is not likely to be a mental ability, a power, a faculty, but which can be inherited and can be attacked by the causal factors of the psychoses is probably of a definite neurological nature.

It may be well to point out here the conditions which the cognitive factor in the psychoses must satisfy if it can ever be measured directly.

Using the battery of tests that have given us our correlations (or a similar battery *mutatis mutandis*) the measure of the general factor must, within the limits of sampling:

(1) Correlate with Reasoning, .853; Perception, .720; Memory Span, .583; Rate of Forgetting, .481; Shut-in, .425. (Cf. Table I.)

(2) Correlate with the group weighted and used as a battery, .914.

(3) When an attempt is made to treat the measure of the general factor as a specific element and tetrad differences are calculated a number of them will then be larger than the limits allowed for errors of sampling and the correlations of the variable with *G* partialled out will no longer approach to zero.

If the cognitive general factor is neurological in nature it is likely that the other general factors are also neurological rather than mental. The nervous system has more than one general property. It may be that some of these general factors will prove to be positive and negative variations of the same quantity. This may be the case in our catatonic and uninhibited groups and in the

partial correlation becomes indeterminate $\frac{0}{0}$. Kelley derives the tetrad equation

in an ingenious fashion from the fundamental product moment formula (cf. Cross Words in the Mind of Man, pp. 35, 38, 46 ff.). His proof assumes there is no correlation between any of the specifics and the general factor. If, however, the general factor were one of the specifics, or two or them or their sum total, this fundamental assumption is violated and the tetrad equation would not hold.

depressions and euphoric excitements.* It is a difficult matter from the purely statistical point of view to say that the general factors present in any two groups are the same or different, † because of the fact that though the two groups may have the same general factor, when an attempt is made to combine them, high tetrad differences are found which may be due either (a) to two general factors, or (b) to the overlapping of the specific factors.

For the present we suggest from inspection of the specific traits that: (1) There is a cognitive general factor; (2) there is a catatonic general factor which may be the negative phase of the uninhibited or kinetic factor; (3) there is a manic factor which for reasons alleged in the legend to Table 8 is generic having two forms, the euphoric and non-euphoric; (4) there is a factor underlying the delusional hallucinatory condition; (5) there is a constitutional hereditary factor manifesting itself as depression. This is not the cognitive factor with which it has a negative correlation. But probably an inheritable defect of the nervous system which does not affect cognition but weakens emotional control, or actually increases native emotivity.

Let us for a moment consider Table 8, in order to gain some information concerning the relationship between the *dementia præcox* group and manic-depressive insanity. The retarded depression and the constitutional hereditary depression have with each other a high significant correlation. This may be because there is one and the same general factor underlying both. The correlations of the depressions, however, with the "præcox" general factor or factors (Nos. III-VI) are not all zero. But they are all negative and though two are rather low (.046 and .019) some are signifi-

* A word of explanation will be helpful in understanding some of the correlations in Table 8. In recording our data some stand had to be taken about conditions that showed reversals of the psychotic picture. It was decided that reversals separated by genuine recovery would be recorded only in one phase or the other. Reversals not separated by complete recovery would be averaged and the symptoms of both phases recorded. Thus there is a positive correlation between the uninhibited and the catatonic, but a negative between euphoric mania and depression.

† A technique is available (an improvement on Kelley's given in *Cross Words in the Mind of Man*, p. 69 ff.), but we have not yet applied it to our data. Cf. McDonough, *Sister Rosa: Studies in Psychology and Psychiatry*, Baltimore, II, 2: 186, 1929.

cantly high.* This, must mean that *præcox* psychoses are in some way intimately associated with the manic-depressive disorders. For some reason, one condition excludes the other. This is a very important fact in the interpretation of these conditions; but we have not been able to discover why these two conditions should be in contraposition to each other rather than simply lacking in any relationship.

SUMMARY.

(1) A technique, based upon Spearman's tetrad difference criterion, has been evolved for the empirical determination of the syndromes of psychiatry.

(2) This technique has given in a purely empirical manner eight syndromes and their constituent symptoms.

(1) The Syndrome of Cognitive Defect which is positively related to *præcox* conditions but, negatively, to manic-depressive.

(2) The Catatonic Syndrome, probably the inhibitory phase of *dementia præcox*.

(3) The Uninhibited or Kinetic Syndrome, probably the excited phase of *dementia præcox*.

(4) The Non-Euphoric Manic Syndrome, which is probably also a phase or form of *dementia præcox*.

(5) The Euphoric Manic Syndrome, which is probably the underlying condition in the manic form of manic-depressive insanity.

(6) The Delusional Hallucinatory Syndrome, also a factor in *dementia præcox*.

(7) The Syndrome of Constitutional Hereditary Depression, which indicates an hereditary factor that attacks the controlling mechanism of emotional life or heightens emotivity, but leaves the cognitive general factor untouched.

(8) The Syndrome of Retarded Depression, which is not as yet clearly differentiated.

* Thus, for instance, the probable error for the correlation ($- .415$) between retarded depression and uninhibited (364 cases) is .081 about 5 times the probable error; that for the correlation ($-.439$) between constitutional hereditary depression and cognitive defect (185 cases) is .085.

(3) Reasons are given for regarding these syndromes as neurological defects of a definite nature.

(4) The measures used in the determination of the general factor can now be applied to the diagnosis of the above syndromes. The development of a procedure for doing this in a definite and simple manner is now in progress.

BIBLIOGRAPHY.

1. Studies in Psychology and Psychiatry, 2, No. 2, 1929.
2. Moore, Thomas: Psychol. Mon., Princeton, 27, 301, 1919.
3. This recalls Wundt's tridimensional theory of feeling which maintains that the simple feelings pass over to their opposites through a zero point of indifference. Cf. Wundt, Wilhelm, *Gründzüge der physiologischen Psychologie*, 5th Ed., 1902, pp. 337 ff.
4. Cf. on this, *e. g.*, Handbook of Mathematical Statistics, ed. by H. L. Rietz, 1924, p. 72 ff.
5. For example, in the appendix to the work of Rietz above cited.
6. Cf. hereon. Spearman, Charles: *The Abilities of Man*, 1927; Kelley, Truman Lee: *Cross Roads in the Mind of Man*, Stanford, 1928.
7. Dodd, Stuart: *Jour. of Ed. Psychol.*, 19: 217, 1928.
8. McDonough, Sister Rosa: *Studies in Psychol. and Psych.*, II, No. 4, 178, 1929.
9. See the work of Morrison, B. M. Univ. of Calif. Publ. in Psychol., 3: 73, 1924, which shows that with morons emotionality is negatively correlated with the degree of mental defect so that the deeper the degree of mental defect the more shallow the intellectual life of the patient. Cf., also, Pages, L.: *Affectivité et intelligence. Étude psychopathologique*, Paris, Alcan, 1926. Cf., also, discussion Moore, Thomas: *Dynamic Psychology*, 101-115, 1924.
10. The Lange-James theory of the emotions can no longer be taken seriously. Cf. T. V. Moore *Dynamic Psychology*, Part III, Ch. ii. For the opinion of a Neurologist cf. Wilson, S. A. Kinnier: *J. Neurol. and Psychopath.*, 4: 299, 1924.
11. Travis, L. E., and Hunter, T. A.: *J. of Exp. Psychol.*, 11: 342, 1928.

POST PROHIBITION ALCOHOLIC PSYCHOSES IN NEW YORK STATE.*

By WILLIAM C. GARVIN, M. D.,

Medical Superintendent, Binghamton State Hospital, Binghamton, New York.

One of the outstanding topics of discussion in the daily press, magazine articles and among our citizens, is the alcoholic question. No popular subject has been accompanied by such an intense individual and group emotional reaction as this self-same topic. It was the dominant feature in our last Presidential campaign. It is the cause *par excellence* of hundreds of thousands of interested men and women who have devoted their time, social and political influence, and emotional energy in endeavoring to further the cause of prohibition, to uphold the present laws and to secure the passage of more drastic ones to enforce the Eighteenth Amendment and the Volstead Act. Moreover, the liquor question has practically become a part of the creed of certain religious sects, who have perfected a militant organization, supported by ample funds, in order to arouse public opinion in support of the existing statutes, to prevent congressional action in the matter of a more liberal interpretation, and to oppose the repeal of the Volstead Act. All this notwithstanding the fact that prohibition has been made a law of our land, having been incorporated into the Constitution of the United States by the mandate of the legislative bodies of 46 of 48 of our states.

It is a popular belief that national prohibition was "put over" solely as a war measure. This opinion is inconsistent with the facts. The movement against alcohol had been gathering strength for a number of decades. At first a few small communities in a state who were favorable to local option would go dry. After a time an additional number of sections would also go dry. When a sufficient number of communities in a state went dry and the number of its citizens were powerful enough to influence legislation,

* Read at the eighty-fifth annual meeting of The American Psychiatric Association, Atlanta, Ga., May 14, 15, 16, 17, 1929.

the entire state would go dry. At the time the Eighteenth Amendment was adopted a large number of the states had passed laws in favor of prohibition, showing that the sentiment of the people had been gradually crystallizing against alcohol.

For years before the passage of the Eighteenth Amendment, industry, the railroads and large employers of labor had placed their taboo upon the alcoholic. The merchant had also seen the light. The practice of having salesmen wine and dine prospective customers so as to secure an order for goods was becoming a thing of the past. Modern business methods were concerned only with price, quality, service and the ability of the customer to meet his obligations.

Those interested in public health and in human betterment were for prohibition, or at least in favor of some method of drastic restriction. The public was becoming educated to the evil effects of the abuse of alcohol, and remarkable progress was being made in the direction of temperance. Furthermore, certain church groups were actively engaged in an effort to pass a national prohibition law.

Thus it will be seen that the movement to restrict the manufacture and sale of alcohol was not a spontaneous affair, but one of slow growth, with economic, moral, societal, criminal, political, mental and physical aspects.

Our entrance in the Great War was accompanied by an emotional exaltation and spirit of sacrifice unparalleled in our history. The great bulk of our citizens cooperated in the effort to save and conserve foodstuffs and raw materials to feed our troops and allies. The manufacture of liquor from grain and other products was curtailed. Governmental restrictions were imposed on the manufacture of alcohol so that it would be available for war purposes. This restriction, together with the spirit of sacrifice displayed by our people, resulted in diminution of supply and the country in general drank less liquor. A number of foreign countries had restricted the manufacture and sale of intoxicating beverages during the war.

The psychological moment had arrived for the radical prohibitionists to commit the entire country to prohibition. Our people, in general, had become disgusted with the Bourbonism of the distiller, brewer, saloon and dive keeper, rotten politics, prostitution and vice, which always accompanies liquor traffic. Thousands,

while not favoring absolute prohibition, voted for the Eighteenth Amendment, believing it might put an end to such intolerable conditions.

The Eighteenth Amendment was passed on January 16, 1920, and the Volstead Act was enacted the following day, over the veto of President Wilson. There was great rejoicing throughout the country among the protagonists of absolute prohibition. As a people we are inclined to emotional crises, sentimentality and to believe that the passage of a law is all that is necessary to remedy conditions to bring about human betterment. The aftermath following the end of the war and the conclusion of peace, the rise of the bootlegger and conditions with respect to liquor traffic at present, is familiar to all. We, as physicians know, and most of our friends are aware of the fact, that there is scarcely a village, town or city in our land where one cannot purchase illicit liquor if one has the price. The fact is that there exists in the country at present what practically amounts to local option. Where the preponderant sentiment in a community is for the suppression of traffic in liquor, that section is fairly dry; where the majority holds the opposite view, it is wringing wet. Where the wets are in the majority the law is openly flaunted and convictions for violation of the prohibition laws are difficult to secure. More drastic laws have recently been promulgated, in order to punish more effectively violators of the national enactments. Whether they will prove successful remains to be seen, but it is highly probable that those individuals who have been accustomed to drink liquor and have the price to purchase it, will, for the most part, continue to do so. When the profit is sufficiently attractive the illegal seller of illicit liquor will take a chance, and the purchaser will salve his conscience, if he has any scruples, by various methods of rationalization familiar to all.

As psychiatrists, we are interested in the liquor question from many standpoints. Psychiatry to-day does not confine its scope to the insane, feeble-minded and epileptic as formerly. We are interested in the individual, his mental reactions and behavior, from infancy to old age. We are vitally interested in the alcoholic question by reason of its social, economic relationships, including dependency, poverty, immorality, vice, delinquency, crime, physical disease, mental disease, epilepsy and mental defect. Physicians

as a group are concerned with the prohibition laws by reason of the limitations set by the federal laws respecting prescribing liquor in hospital and private practice and the burdensome regulations governing such dispensing.

With this preamble I will proceed to the main theme of this paper—Post-Prohibition Alcoholic Psychosis in New York State.

TABLE I.

FIRST ADMISSIONS WITH ALCOHOLIC PSYCHOSES, CIVIL STATE HOSPITALS OF NEW YORK, 1909-1928.

Year.	Number.			Per cent of all first admissions.		
	Males.	Females.	Total.	Males.	Females.	Total.
1909.....	433	128	561	15.6	5.8	10.8
1910.....	452	131	583	15.3	5.0	10.5
1911.....	444	147	591	14.7	5.5	10.4
1912.....	434	131	565	14.4	4.8	9.8
1913.....	438	134	572	13.7	4.7	9.4
1914.....	348	116	464	10.4	3.6	7.4
1915.....	255	90	345	7.8	3.1	5.6
1916 *.....	215	82	297	8.4	3.5	6.1
1917.....	437	157	594	12.1	4.8	8.6
1918.....	257	97	354	7.3	3.0	5.2
1919.....	204	65	269	5.8	2.0	4.0
1920.....	90	32	122	2.7	1.0	1.9
1921.....	167	26	193	4.6	0.8	2.8
1922.....	194	32	226	5.1	1.0	3.2
1923.....	220	56	276	6.1	1.7	4.0
1924.....	302	71	373	8.2	2.2	5.4
1925.....	341	81	422	8.8	2.3	5.7
1926.....	333	89	422	8.4	2.7	5.8
1927.....	440	114	554	10.1	3.2	7.0
1928.....	430	79	509	9.1	2.0	5.9

* Nine months.

The statistics are taken from the annual reports of the New York State Department of Mental Hygiene, and were prepared by Dr. Horatio M. Pollock, statistician. I also wish to acknowledge my indebtedness to Dr. Pollock for making use of statistical data and comments on the alcoholic psychoses published in several contributions in *Mental Hygiene*:

Table I shows first admissions of alcoholic psychoses to the New York State civil hospitals for the years 1909-1928. It will be seen that up to 1915 there was a steady decline in first admissions. I might state here that the New York State Mental Hygiene Law

does not permit us to accept as patients, ordinary cases of alcoholic intoxication; patients must present psychotic symptoms at the time of admission. There was a slight rise in the curve in 1916 (a nine-month period) *viz.*, 6.1 per cent. In 1917 occurred a further rise, *viz.*, 8.6 per cent. Prosperity at that time was rampant and money flowed freely. We entered the World War on April 7, 1917. The war crises with its concomitant excitement, easy money, and increased use of intoxicants doubtless accounts for this rise, which is the highest up to 1928. The lowest point in the curve, 1.9 per cent, was reached in 1920. The manufacture of liquor had been curtailed, our people cooperated in every way to conserve our food and economic resources; the sale of liquor was carefully watched and drinking became less general. Bootlegging had not yet become well organized.

The end of the war and the return of our troops to civil life, the passing of emotional enthusiasm incident to the strife, the resumption of more normal habits of living, and the failure of many of our citizens to obey the provisions of the Eighteenth Amendment and the Volstead Act, also the development of bootlegging on a wholesale scale, making liquor more accessible, have occasioned a steady increase in the number of first admissions. In 1927 they reached a total of 7 per cent. In 1928 the rate dropped to 5.9 per cent. What the future will reveal no one can say. The effect of the drastic Jones Law remains to be seen. In this connection the psychological attitude of the individual and group must be taken into consideration. The fact remains that when there is less drinking, there are fewer cases of alcoholic admissions than formerly. It is perfectly true that the figures show a decrease of admissions from 10.8 per cent in 1909 to 5.9 per cent in 1928. This represents a percentage drop of 45 per cent, but it should be borne in mind that the rate had already begun to decline noticeably before the prohibition enactment went into effect, due to less drinking in general, on account of the education of the public to the ill-effects of abuse of alcohol, local option and state prohibition. No doubt the prohibition amendment was passed a bit too prematurely, but eventually it will become accepted as the law of our land by the great bulk of our citizens.

Undoubtedly the effect of dry laws upon the younger generation will, in the course of time, be to their material benefit mentally,

physically, morally and economically. Social conditions have materially changed since pre-war days; wages and salaries have been increased; standards of living have been raised to a high level. The saloon has disappeared; the "speak-easy" is still with us, but is, as a rule, mainly patronized by the habitual user of alcohol. There are many more healthy outlets for young men at present, as the auto, moving pictures, sports, radio, etc. As for the drinking among our young people, it is highly probable that after a certain amount

TABLE II.

INTEMPERATE USERS OF ALCOHOL AMONG FIRST ADMISSIONS, 1911-1927.

Fiscal year ending.	Number.			Per cent of total first admissions.		
	Males.	Females.	Total.	Males.	Females.	Total.
1911.....	1082	302	1384	35.9	11.2	24.3
1912.....	1097	273	1370	36.5	10.0	23.8
1913.....	1103	318	1421	34.6	11.1	23.5
1914.....	1027	258	1285	30.8	8.8	20.5
1915.....	939	225	1164	28.8	7.5	18.7
1916.....	725	182	907	28.2	7.8	18.5
1917.....	1152	300	1452	32.0	9.2	21.1
1918.....	851	253	1194	24.1	7.7	16.2
1919.....	804	161	965	22.8	4.9	14.2
1920.....	684	119	803	20.3	3.7	12.2
1921.....	691	131	822	18.9	4.0	11.8
1922.....	757	122	879	21.1	3.8	12.5
1923.....	756	130	886	20.9	4.0	12.8
1924.....	842	165	1007	22.8	5.1	14.5
1925.....	813	167	980	21.0	4.5	13.2
1926.....	832	170	1002	21.0	5.1	13.7
1927.....	1050	211	1261	24.1	5.9	15.9

of adolescent flurry of drinking, they will eventually settle down and become stable, temperate citizens, like the majority of our people.

The statistics (Table II) of the State Department of Mental Hygiene, prepared by Dr. Pollock, also show another interesting fact, *viz.*, that there was a considerable decline in the excessive use of alcohol among all first admissions to the New York State Hospitals prior to prohibition. (*Vide*, "Decline of Alcohol and Drugs as Causes of Mental Disease." *Mental Hygiene*, January, 1921.)

Of 1384 first admissions in 1911, 24.3 per cent were recorded as having been intemperate in their use of alcohol. From this year

on there was a steady decline, except in 1917, when the rate rose to 21.1 per cent. The lowest point was reached in 1920; in this year 803 patients, or 12.2 per cent, used alcohol intemperately. In view of the fact that the Eighteenth Amendment and the Volstead Act did not go into effect until January, 1920, the lowering of the rate of intemperate drinking among all first admissions and the reduced rate of alcoholic psychotic first admissions had nothing to do with any national prohibition laws.

Dr. Pollock's statistics show that, except in cases of pathological intoxication, the average period of drinking for men was 22.2 per

TABLE III.
RACES OF ALCOHOLIC FIRST ADMISSIONS—1914-1927.

Year.	Unas.	Mixed.	Irish.	Italian.	Scan.	Slav.	Heb.	African.	German.	English.	Total.
1914	10	107	178	14	12	19	2	10	55	36	443
1915	38	50	121	13	9	16	1	8	52	19	327
1916	24	27	121	10	7	19	3	11	37	19	278
1917	42	86	239	13	14	44	3	22	5	24	492
1918	14	72	125	11	9	40	2	14	38	13	338
1919	15	48	90	13	5	26	3	10	32	8	250
1920	6	28	47	4	2	13	0	2	12	5	119
1921	2	40	71	11	6	17	1	6	21	5	180
1922	6	37	74	18	6	28	5	6	23	9	212
1923	5	45	80	16	14	44	2	7	25	12	250
1924	8	80	110	26	15	43	6	28	23	13	352
1925	9	82	145	29	13	49	4	14	38	11	394
1926	13	90	127	23	11	57	2	16	39	13	391
1927	12	115	164	27	13	76	10	46	36	19	518
Total	204	907	1692	228	136	491	44	200	436	206	4544

cent years, and for women 16.4 per cent years. He found that over one-half of the new cases reached the hospitals between the ages of 35 and 50 years, and that women with alcoholic psychoses entered the hospital at a later age than men. A discussion of the psychodynamics of the alcoholic psychoses is beyond the scope of this paper.

One of the interesting problems in connection with the alcoholic psychoses is its incidence in the racial groups who come to us as patients. We have many diverse nationalities in our land; some drink but little; others more temperately, while still others drink immoderately. The statistics (Table III) from the annual reports

of the New York State Department of Mental Hygiene may prove of interest.

It will be observed that the Irish for years have furnished the largest number of first admissions throughout the entire period. There has been a decided increase in the number of Slavonic admissions, no doubt due to the increased number of this race in centers of population; the same holds true of the negroes, who have migrated from the south to New York City in large numbers in recent years. There is also an increase in the number of Italian admissions. The lowest admission rate is among the Hebrews, who are temperate in their use of alcohol.

Let us review the statistics of first admissions of alcoholic psychoses to the Binghamton State Hospital for the years 1909 to 1928. Our district embraces nine counties along the southern tier of New York State. The total population of the nine counties is approximately 460,000. Binghamton, Johnson City, Endicott and Port Dickinson are all contiguous towns, with a total population of about 125,000. In this section are a large number of shoe factories employing considerable foreign population, many of them Slavs. Among other towns in our district is Elmira 40,000, and in addition several other cities of 15,000; the balance of the district is rural in character. A large majority of our alcoholic admissions come from our urban centers. But few women with alcoholic psychosis are admitted from our district.

During the period from 1909 to 1928, there were 209 first admissions of alcoholic psychoses; 185 men, and 24 women. In 1909 and 1910 sub-types were not recorded.

From 1911 to 1928, the specific psychotic groups were as follows:

Acute and chronic paranoid states.....	7
Chronic deterioration	25
Korsakoff syndrome	21
Delirium tremens	19
Acute and chronic paranoid states.....	7
Other types	14
Non-specified types admitted in 1909-1910.....	32
	—
Total	209

During the period from 1909 to 1928, a total of 4531 first admissions were received, the alcoholic group representing an admission

rate of 4.6 per cent. The lowest rate of alcoholic admissions was in 1920, when only one case was admitted, the highest number admitted was in 1927, *viz.*, 23 patients. The outstanding type admitted prior to and since prohibition has been the acute alcoholic hallucinosis. We rarely encounter pathological intoxication; Korsakoff's psychosis has been less numerous since 1920, and, moreover, the mental symptoms and the polyneuritis have been much less pronounced since the war. Recovery is also much more complete.

With respect to the acute alcoholic hallucinosis, we also note the following differences since the war: Patients, owing to the character and quantity of the liquor imbibed, appear more toxic on admission; there is a greater degree of physical prostration than formerly; many of them have to be kept in bed and in order to relieve the toxic and dried-out state, hypodermoclysis of normal saline solution and intravenous injections of solutions of glucose often have to be administered; the sensorium is more clouded; confusion and disorientation are often present; there are more delirious admixtures; hallucinations are often dream-like in character and are frequently of the combined type, *i. e.*, both visual and auditory in nature; they have a hazy recollection of the acute phase of the disease. This is in striking contrast to the acute alcoholic hallucinant observed prior to the war, who could ordinarily give quite an excellent account of all that transpired during the acute stage. Recovery is more gradual now-a-days. While the onset is usually fairly acute, the auditory hallucinations are not so outstanding as formerly, and there is a great deal less systematization. Moreover, on account of the prostration, toxic delirious state and clouding of the sensorium, there is less acute anxiety and fear. Hallucinations, when they do appear, are usually of a threatening, persecutory, defamatory character, and in men there is often observed hallucinations of a homosexual nature, while accusations of immorality and infidelity are common in women.

The diagnosis between acute alcoholic hallucinosis and dementia præcox is often not possible for a time, especially in the absence of a good anamnesis and the presence of language difficulties. A good life history of the patient secured from relatives or others, and also from the patient himself; accurate knowledge concerning the type of personality of the patient, with exact information respecting the frequency and amount of alcohol imbibed; information regard-

ing previous attacks; reliable data concerning the acuteness of the psychotic symptoms, are all of value in establishing a diagnosis, especially if the characteristic features of the acute alcoholic hallucinosis, as described by Bonhoeffer, are borne in mind.

As a rule, the dementia præcox is not a chronic drinker. Individuals with schizoid make-up do sometimes drink and develop what appears to be an acute hallucinosis; in many such instances we have to do with the onset of dementia præcox. These patients show an absence of adequate emotional response to the hallucinatory and delusional formations; as a rule, there is not the same degree of anxiety, restlessness and fear; in other words, they accept the situation to a more or less extent. In the alcoholic hallucinant his anxiety, fear, restlessness, suicidal and homicidal tendencies all show the conflict which is present in his mind and his struggles against it. Furthermore, the personality of the alcoholic hallucinant is different from that of the dementia præcox; he is not the queer, shy, seclusive person we find in cases of dementia præcox, but is generally regarded as a pretty good sort of a fellow, liking the company of his own kind. The homosexual character of the psychic material is often quite noticeable.

As to the reason for the changes in the alcoholic psychotic picture which I have mentioned above, I am of the opinion it is due to the fact that there is not so much continuous and excessive drinking among alcoholics as formerly, as it is too costly; moreover, the quality has deteriorated and is more poisonous in character. The chronic alcoholic does not drink beer any more because he cannot get it. He is, as a rule, not an habitual wine drinker, as this fails to have the requisite "kick." He drinks excessively what he has the funds to purchase, and this is chiefly the cheap so-called whiskey and gin. Government analyses show that most of the hard liquors furnished by the bootleggers are obtained from re-distilling methyl spirits, and that it is practically impossible for the ordinary bootlegger distiller to remove all the noxious ingredients. Our patients are not able to afford the higher grades of liquor purchasable by the rich. The result is that many of them when they come to us appear poisoned, and during the acute period of the disease are generally very much confused, and when they clear up are unable to give a clear retrospective account of their experiences. Practically all of

them give a history of obtaining liquor from bootleggers, and having indulged in recent excesses. Imaginations then appear and after that there is a great deal of confusion as to what happened subsequently. Many of them are picked up on the street on account of acting queerly, and taken to jail; others act peculiarly in their homes and are committed direct to the hospital. It is only after several days in the hospital that the sensorium begins to clear up and they realize where they are. We group a considerable number of these cases as acute alcoholic hallucinosis on account of prominence of the auditory hallucinations, with varying degrees of anxiety or fear reaction developing in connection with recent alcoholic excesses; in fact, in the majority of instances, we are dealing with a more or less delirious state.

A surprising number of recoveries take place among all types of alcoholic psychoses, despite the quality of the liquor drunk. This is probably due to the fact that they are not able to drink liquor continuously as chronic alcoholics did in former days, and, therefore, they are not so chronically poisoned.

Dr. Menas S. Gregory, Director, Psychopathic Department, Bellevue Hospital, New York City, writes me that very few alcoholic psychoses are committed by him to the state hospitals receiving patients from Bellevue, as most of them are kept in the Psychopathic Department long enough to recover. He remarks:

Speaking again for Bellevue Hospital, after prohibition went into effect, alcoholism changed from a chronic condition to an acute poisoning. Since the individual could not secure liquor whenever desired, he drank as much as possible when the opportunity offered. Thus chronic alcoholism almost disappeared and the drinking consisted of acute poisoning, due mostly to the large amount of liquor consumed and to a very small extent to the poor quality. When admitted to the hospital they were almost uniformly in a state of coma, undoubtedly due to excessive drinking rather than due to the quality of the liquor drunk. Another feature is that they do not recover as quickly as formerly owing to the serious toxic condition. The various features of chronic alcoholism, such as cardio-nephritic conditions, peripheral neuritis, alcoholic hallucinosis and alcoholic paranoia, entirely disappeared or became much less. However, during the past few years, as it has become just as easy to acquire liquor as in pre-prohibition days, the phases of chronic alcoholism (as in the pre-prohibition days) are gradually beginning to appear again.

Table IV, showing the admissions to the alcoholic wards of Bellevue for the years 1909-1928, may prove of interest. It will be observed that the admissions reached their low point in 1920, and since then there has been a gradual rise in the curve, as is the case in the New York State Hospitals.

TABLE IV.

ADMISSIONS TO THE ALCOHOLIC WARDS OF BELLEVUE HOSPITAL,
NEW YORK CITY, 1909-1928.

Year.	Men.	Women.	Total.
1909.....	9447
1910.....	8668	2721	11389
1911.....	7111	1058	8169
1912.....	5598	2309	7907
1913.....	6052	1821	7873
1914.....	5778	1864	7642
1915.....	4862	1509	6371
1916.....	7114	2179	9293
1917.....	6443	1767	8210
1918.....	2002	1346	3348
1919.....	2351	605	2956
1920.....	1642	449	2091
1921.....	1931	450	2381
1922.....	3305	778	4083
1923.....	4685	1092	5777
1924.....	5205	1139	6434
1925.....	4880	1055	5935
1926.....	4938	986	5924
1927.....	5520	1027	6547
1928.....	5881	1118	6999

SUMMARY.

A marked decline in first admissions of alcoholic psychoses to the New York State hospitals has taken place during the past two decades.

The lowest admission rate occurred in the fiscal year ending June 30, 1920, *viz.*, 1.9 per cent.

Since 1920 there has been a gradual rise in admissions, the peak of the curve being reached in 1927, *viz.*, 7.0 per cent. In 1928 the rate dropped to 5.9 per cent.

The reduction in the admission rate has been relatively greater among women than among men.

First admissions with alcoholic psychoses come principally from urban districts.

The rate of admissions with alcoholic psychoses is greater among the foreign born than among the native white born.

The two races in which alcoholic psychoses are most prevalent are the Irish and the Slavonic races.

The rate is relatively higher among Negroes than among the native white population.

Alcoholic psychoses among the Hebrew race is comparatively rare.

Alcoholic psychoses, as a rule, occur principally in middle age, following years of intemperate use of alcohol. The acute phase generally develops in connection with recent excesses.

There has been a general reduction in the intemperate use of alcohol among all first admissions to the New York State hospitals from 1911 to 1927.

Acute hallucinosis is the outstanding type of alcoholic psychosis admitted to the Binghamton State Hospital.

There has been observed a change in the symptomatic picture as compared with the pre-war type, in that the patients appear more toxic and prostrated. There is often confusion, disorientation, combined hallucinations and delirious admixtures during the acute phase.

Recovery in the more severe cases is slower than in pre-war days.

Korsakow's psychosis has decreased in frequency since the war, and the mental symptoms and polyneuritis are much less severe than formerly. Permanent mental and physical residuals are less common, and recoveries are more frequent than in the pre-war period.

DISCUSSION.

DR. CHARLES ENGLANDER (Newark, N. J.).—I have two questions I would like to ask Dr. Garvin. One is, can he give us any data as to the occurrence of these alcoholic psychoses in the age groups up to the age of 30. There has been so much said about the younger folks drinking more since prohibition that I think it would be of some interest to have those figures if they are available.

The other is whether the quality of the alcohol being different or worse is responsible for these pictures. I am inclined to think that we might be justified in calling some of these so-called alcoholic psychoses toxic psychoses rather

than placing them in the alcoholic toxic group. When the prohibition movement first was actively being enforced, there were a great many instances of admissions of peculiar types of psychoses, and I think some work was done in the vicinity of New York City. I know some was done in Newark, in which it was found many of these alcoholics at the time were suffering from acute accumulation of aldehydes and other chemicals. If that is true, in contradistinction to the purer types of psychoses we saw formerly, certainly we ought not to ascribe these psychoses, we ought not to place them in the purely alcohol group.

The other thing is this: are we justified in saying that prohibition is responsible for the increase? Is not it more likely true that the actual increase is due to the fact that the Irish race, the Germans and the Slavs are more prone to go to the trouble of making home-brew and as a result their manufactured new product contains these toxic substances. Is not it again true that the conditions that we are seeing are the toxic type of thing, chemical in nature, other than alcohol? I do not think we can altogether say that delirium tremens is disappearing or is less prevalent.

From state hospital figures, we might be led to believe that this is true. It is also true the state hospitals and county hospitals get fewer of those cases. In the large general hospitals, we still see a great many of the delirium tremens cases and many of the acute hallucinatory things. I do not believe that in the general hospitals the types of psychoses we are seeing are a different type of picture, as we might be led to think from some state hospital experiences.

DR. NEIL A. DAYTON (Boston, Mass.).—I would like to draw Dr. Garvin's attention to one point. In stating that the Slavonic and Irish races are particularly prone to alcoholic psychoses, he has failed to consider the proportions of these races in New York State. He is hardly justified in his conclusions when they are based on numbers of cases alone. In his statements in reference to the racial distribution of alcoholic psychoses and the increases that have taken place in these races over the past 10 years, he must consider other factors. To get an unbiased viewpoint of this matter, we must take into consideration the proportion of these races in the population and also the increases in these races that have taken place over the same period of years which was used in discussing the psychoses. If conclusions are to be drawn from the material of this paper the figures must be corrected for population. One cannot simply attach psychoses to races without subjecting the material to a little finer analysis.

DR. ADOLF MEYER.—I cannot help expressing an impression that seems to me of considerable importance in this whole problem; namely, that we must not deal with this question purely on a statistical basis. Statistics are of great help and they are furnishing us a background. After all, one of the important things is to get at the essence of things, and, unfortunately, we are at present, or have been during the last 10 years nearly, in a period where we have ceased

to think in terms of the alcohol problem and think too much of the prohibition problem or the anti-prohibition problem, or whatever we want to call it. We cannot say that the present conditions are due to prohibition. I think they are to a very large extent due to the non-prohibition. I feel that we have got to recognize that we as psychiatrists have two problems on hand, certainly we have the economic problem of how many patients are being produced under the present civil war concerning this matter or the present anarchy, the remonstrance process. I am pretty sure that a very large percentage of what constitutes the problem at the present time is a remonstrance problem. When we come to the question of the alcohol itself, I made some statistics in Worcester and Dr. Kirby did it on his own account on Ward's Island. They were done on this ground, that the number of patients admitted from various nationalities and groups were to be studied for the relative proportion among them of the cases that actually presented alcoholic psychoses and not only psychoses in which alcohol played perhaps a rôle.

Before prohibition was at all spoken of, when one had local option in Massachusetts, there was a very interesting differentiation, so that, for instance, of the men born in Ireland and admitted to the Worcester State Hospital for any sort of psychoses, 50 per cent had very definitely alcoholic psychoses; whereas, of the Massachusetts born, the non-Irish people, 9 per cent had alcoholic psychoses, and from among the Hebrew patients of that group, there was a small number, there was less than 1 per cent. So that we see the alcohol problem is one of groups and ought to be studied from a sociological and economic point of view to quite an extent. We should then recognize what a tremendous responsibility rests upon the individual and also upon those who are working on this question when we approach what we are doing to the Negro by our remonstrance propaganda. I do not think any one of you would take me as a propagandist or as a fanatic in these matters. I simply would say that as physicians and psychiatrists, it would be a very desirable thing for us to make a clear distinction between what we want to do about remonstrance and what we want to do about the alcohol problem. The alcohol problem, to my mind, is to a very large extent one of social groups and the American people prove it to-day. I think it is very largely a social issue and an issue of remonstrance groups.

With regard to the quality of alcohol and changes of psychoses, I am not so sure that we can say so very much. I only should like to add that we deal not only with a local problem. Switzerland, for instance, has no prohibition issue; it has alcohol legislation and efforts, but there the schnapps is increasing to a point of becoming a national calamity. Not that there is sentimentality involved there and purism or anything of the sort; it is simply a fact that unfortunately, until it is possible to use the surplus of fruits for purposes that will bring a little more money than schnapps will, we shall have an alcohol problem.

DR. HORATIO M. POLLOCK (Albany, N. Y.).—Mr. Chairman, I wish to call attention to the fact that the results shown for New York State by Dr. Garvin are very similar to those obtained by similar statistical studies in other

states. For the entire nation we have very poor comparative statistics of the alcoholic group. In 1910 the Federal Census Bureau attempted to get the percentage of alcoholic cases among all admissions. It was found that the percentage was approximately 10 in that year; in 1922 a similar attempt was made and the data compiled showed that the percentage of alcoholic cases among all admissions had dropped to 3. In Massachusetts, Illinois and other states, where fairly good comparative records are available, we find a trend similar to that in New York State.

In regard to the prevalence of alcoholic psychoses in the various races, unfortunately we have no general census giving the distribution of the population by races, but we do have the distribution by nativity which is compiled by the Federal Census Bureau every 10 years. In 1920, we calculated rates for New York State based on such distribution and found a very high rate of alcoholic mental disease among the Irish of both sexes. I think it is also true that the Irish in their own country have a high rate of alcoholic insanity.

From our statistics it appears that the problem of the alcoholic psychoses is most serious among the foreign-born in cities.

DR. W. C. GARVIN (Binghamton, N. Y.).—As to the appearance of alcoholic psychoses before 30, we meet with them rather infrequently. As to their occurrence also in the jazz age, I have rarely encountered them in state hospital practice. They are usually taken to private or general hospitals. The probabilities are that the morning after, they have had about enough liquor and are quite willing to lay off it for some time to come, unless they are more or less feeble-minded or psychopathic.

Far be it from me to say alcohol causes a mental condition *per se*; I do not believe that for a minute. I believe that all the material which appears in the mental content of the clinical picture, or expressed by the actions of the patient, is derived mainly from the psyche of the individual and not from the alcohol. It is also a question in my mind whether alcohol stimulates the auditory approaches, as has been claimed, or any other area in the brain preponderantly or disproportionately. I am a firm believer in the psychic origin of alcoholic mental disease, admitting of course, the influence of physical causes seen in Korsakoff condition and chronic alcoholic poisoning where we have the definite brain pathology, or other concomitant physical conditions.

The statistical basis, of course, is only one method of approach in psychiatric-sociological problems. In the time allotted me, it is impossible to say much as to the personality of the alcoholic psychotic. Such papers, I believe, should be reserved for a very small group of cases that can be thoroughly and intensively studied.

Notes and Comment.

THE FUTURE OF THE ASSOCIATION.*—As I near the end of my term as President, I naturally think of those matters which are likely to be of importance to the Association in the future, and which will be intrusted to new and capable hands.

A five-year experience in different offices induces me to recommend to the Association what the wise constitution makers arranged—the use of the Council as a central and co-ordinating body. Officers and committees come and go, the Council goes on forever. It is small enough to save the Association much time. One chief duty rests with the Association—to see that the Council is truly representative. The Association should once in a while turn down a recommendation of the Council just to keep it in its place, but I very much doubt the advisability of the Association's considering any matter without a recommendation of the Council for or against it.

Of immediate concern to the Association and Council should be the experiment with the program to be tried this year under the unusual conditions at Washington. After this program is carried out, how many ideas about our usual program will it suggest? Perhaps some of the 1930 methods may be kept for later meetings.

In Association management I hope that some way will be found to make committee appointments more adequate to the situation. I was one of those who believed that wholly new committees each year made for discontinuity of work and reports. But the new scheme of five-year appointments has disadvantages as time goes on. Any president and council loses influence on the committee as only one-fifth can be appointed in the year. Chairmanships form

* The following has been contributed to the JOURNAL by Dr. Bond by request of the Editor. Dr. Bond asks that we write "an introduction which will be an apology for it." No apology is necessary, nor do we think an introduction needed.

We hope Dr. Bond's example will be followed by future Presidents, who may in this manner say things to the members concerning our Association which they may not wish to include in a formal address.

an awkward problem: if a chairman has served five years it is awkward to reappoint him (to make a ten-year term) and awkward not to (and seem to show ingratitude). Chairmen still do most of the work on most committees. Fellows might be ineligible for reappointment on committees. When no report comes from a committee it might automatically be dropped, or reappointed with none of its former members.

A problem which is bound to come up is that of a permanent office for the Association, with a paid (lay?) secretary. Some state medical associations have organized successfully in this way, but it is a question as to whether our Association has the kind of business which would justify the expense. Certainly this matter should be considered in the appointment of a new Editor, because an Editor's work could be relieved in many ways by a paid secretary located in an office where all records would be stored.

The Association can, if it wishes to, move on to some fundamental considerations. One of these is what instruction and what achievements should justify a physician in calling himself a psychiatrist. Closely related is what kind of teaching in psychiatry is needed for the degree of M. D.

And not separated so far from graduate and undergraduate teaching is the question of standardization of mental hospitals, because this implies, as Dr. Meyer says, "the kind of atmosphere and general base line of preparation" which may either encourage or discourage the young man investigating what there is in psychiatry.

There is one way only to approach these fundamentals and this is a constructive way, with emphasis on those steps which will bring commendation to the medical schools and hospitals which take them.

The investigation of the proper relationships of the American Psychiatric Association to other associations in the field of psychiatry—general or special—is being carried on well. Without pressure being used except that which comes from the moral obligation to make each organization efficient, it has been agreed that one association should be absorbed, that another should be consulted as to programs and meeting places, and that others should meet with us for general discussions of co-operative possibilities. Nevertheless it will take initiative in this body to keep it what it now is—a central, rather loose, inclusive, co-ordinating body.

The usual combination of theory and practise will push the Association forward. I hope that every member will formulate a policy, and then very practically keep an eye on the nominating committees and their recommendations as to officers and councillors.

EARL D. BOND.

CENSUS OF INSTITUTIONAL STAFF VACANCIES, ETC.—The JOURNAL invites the cooperation of all hospital superintendents in the effort to secure reliable and complete information concerning the current situation regarding institutional staff. There is being mailed to the recorded head of each institution dealing with mental and nervous patients throughout the United States and Canada a census form in which information is asked as to the proportion of acute and chronic patients dealt with by the hospital; the amount of a physician's time to which patients of each type are regarded as entitled; the number and distribution of present staff; the number of staff vacancies now existing and for which salaries and maintenance are actually available; and the number and distribution of additional staff positions which the superintendent would make serious effort to have authorized and filled if competent psychiatrists were available. The census form also invites a statement as to the extent of medical and psychiatric examination made in the case of each patient; the amount of clinical records kept, and the clerical and related personnel engaged in this work.

Because of the growing interest in some quarters as to the utilization of students from related fields, an inquiry is made as to desirability of assistance in the shape of skilled psychiatric social workers and graduate students in the social sciences who might be available for duty in the shape of intensive study and documentation of some acute mental patients—this under medical supervision.

Finally, a statement is invited as to the number of medical officers engaged in psychiatric teaching, the time spent by each, the character of instruction and the magnitude of the classes which they teach.

It is hoped that preferential consideration may be given to the preparation of this rather extensive census form because there are now some prospects that a concerted and far-reaching attack may soon be made upon the problem of remedying the existing dearth

of personnel for the care of mental patients. The prompt return of accurately completed forms is desired in order that analysis and tabulation of the data may be begun without unnecessary delay and in ample time for the use of the Association committees interested in these problems, before the ensuing Annual Meeting. The returns as such will not be published, but only tables and conclusions derived from the data submitted in them.

DR. C. FLOYD HAVILAND.—The American Psychiatric Association has lost in the death of Dr. Haviland of the Manhattan State Hospital, New York, one of its most active and valuable members.

Two years after joining the staff of the hospital of which he afterwards became Medical Superintendent, as medical interne, Dr. Haviland became in 1899 an associate member.

From the outset he became actively interested in the work of the Association and formed therein many close and enduring friendships. He presented papers which attracted favorable comment and took an active and intelligent and enlightening part in discussion of other papers.

He served on various committees and his position on these was never regarded by him as calling for anything but the best service he could give.

In 1921 he was elected Secretary-Treasurer and was chosen Vice-President in 1924 and President in 1925, presiding at the annual meeting in New York in 1926.

Dr. Haviland's presidential address, which had for its title *Psychiatry and the State*, was published in the *JOURNAL* for July, 1926, as was also a reproduction of his photograph.

In his address he quoted from Huxley as follows: "There is no remedy for the ills of mankind without absolute veracity of thought and action. The resolute facing of the world as it is, with all the garments of make-believe shorn off."

This quotation aptly represents our deceased friend's methods of thought and action. He sought to find the truth, and when found to apply its teachings to the conditions of his fellow men as he found them. For sham and make-believe, for pseudo-science he had no use. A man of broad sympathies he never permitted sentiment to warp calm deliberate judgment or mislead remedial activities.

A tribute to his memory is published elsewhere, but the Editor and his associates desire to record here their profound sympathy for his wife and father and brother as well as their affectionate regard for one who will be long missed by them as well as by those members of our Association who had the good fortune to be brought into intimate relations with one who by his personality compelled their admiration and won their affection.

E. N. B.

DEDICATION OF THE NEW NEW YORK STATE PSYCHIATRIC INSTITUTE AND HOSPITAL IN NEW YORK CITY.—On Tuesday and Wednesday, December 3 and 4, 1929, the new Psychiatric Institute and Hospital at West 168th Street, New York, a part of the University of Columbia-Presbyterian Hospital Medical Center was dedicated with appropriate ceremonies.

At the morning session of Tuesday, Dr. George H. Kirby, Medical Director of the Institute presided, and after a few introductory remarks introduced in order the following speakers: Dr. Frederick W. Parsons, Commissioner of the State Department of Mental Hygiene; Dr. William Darrach, Dean of the College of Physicians and Surgeons, Columbia University; Dr. Walter W. Palmer, Physician to the Presbyterian Hospital; Dr. Frederick Tilney, The New York Neurological Institute; Dr. Nicholas Murray Butler, President of Columbia University; Hon. Herbert H. Lehman, Lieutenant-Governor of the State of New York.

After lunch, papers were read by the following medical gentlemen: Dr. David K. Henderson, University of Glasgow, Scotland; Professor Eugen Bleuler, University of Zurich; Professor Edward Strecker, Jefferson Medical College, Philadelphia, Pa.; Professor Henri Claude, University of Paris.

On Wednesday, during the two sessions, the following gentlemen contributed to the scientific program: Professor Adolf Meyer, Henry Phipps Psychiatric Clinic, Johns Hopkins University; Dr. Wm. A. White, St. Elizabeths Hospital, Washington, D. C.; Professor Ernst Kretschmer, University of Marburg; Dr. Ernest Jones, London, England; Professor William F. Lorenz, University of Wisconsin; Professor C. Macfie Campbell, Harvard University; Professor Walther Spielmeier, University of Munich; Professor Franklin G. Ebaugh, University of Colorado; Professor Constantin Von Economo, University of Vienna.

ANOTHER NAME ADDED TO THE MARTYROLOGY OF PSYCHIATRY.—Dr. Booth E. Miller, Clinical Director of the Harrisburg, Penna., State Hospital, was killed by a patient in the hospital on January 8, 1930.

The circumstances attending the tragic event as far as we are at present informed are as follows:

Dr. Miller was visiting certain wards of the hospital usually under the care of another member of the staff, in that physician's absence. While he was in conversation with a patient another patient came up behind him and drew a razor across Dr. Miller's throat, inflicting wounds from which he died almost immediately.

Whether this patient held any delusions about Dr. Miller we have not ascertained, nor whether he was known to have homicidal tendencies; but at the coroner's inquest it was disclosed that the patient who killed Dr. Miller had obtained in some manner three razors from an acquaintance by threatening to inform the prosecuting attorney that he had killed a man with his automobile. It seems to us that prompt action should be taken against the man who furnished the razors. Dr. Miller's body was taken to California, where his parents live, for burial. We hope shortly to publish a memorial notice of Dr. Miller.

Association and Hospital Notes and News.

THE AMERICAN PSYCHIATRIC ASSOCIATION, OFFICERS AND COMMITTEES 1929-1930:

Earl D. Bond, M. D., President.....Philadelphia, Pa.
Wm. M. English, M. D., Vice President.....Brockville, Ontario.
Robert L. Dixon, M. D., Hon. Vice President.....Wahjamega, Mich.
Clarence O. Cheney, M. D., Secretary-Treasurer.....Poughkeepsie, N. Y.

COUNCILLORS.

FOR THREE YEARS.

Samuel T. Orton, M. D.....New York, N. Y.
Malcolm H. Bliss, M. D.....St. Louis, Mo.
Frank W. Robertson, M. D.....New York, N. Y.
N. W. Owensby, M. D.....Atlanta, Ga.

FOR TWO YEARS.

Adolf Meyer, M. D.....Baltimore, Md.
Henry I. Klopp, M. D.....Allentown, Pa.
Arthur F. Kilbourne, M. D.....Rochester, Minn.
Ross McC. Chapman, M. D.....Towson, Md.

FOR ONE YEAR.

B. M. Hodskins, M. D.....Palmer, Mass.
H. D. Singer, M. D.....Chicago, Ill.
George M. Kline, M. D.....Boston, Mass.
Thomas A. Ratliff, M. D.....Cincinnati, O.

AUDITORS.

FOR THREE YEARS.

Marcus A. Curry, M. D.....Morris Plains, N. J.

FOR TWO YEARS.

F. A. Carmichael, M. D.....Osawatomie, Kans.

FOR ONE YEAR.

L. V. Guthrie, M. D.....Huntington, W. Va.

EXECUTIVE COMMITTEE.

Earl D. Bond, M. D.....	Philadelphia, Pa.
W. M. English, M. D.....	Brockville, Ont.
Clarence O. Cheney, M. D.....	Poughkeepsie, N. Y.
George M. Kline, M. D.....	Boston, Mass.
Henry I. Klopp, M. D.....	Allentown, Penn.

COMMITTEE ON PROGRAM.

FOR FOUR YEARS.

Lawson G. Lowrey, M. D., Chairman 1929-30.....	New York, N. Y.
G. Kirby Collier, M. D., Vice-Chairman 1929-30.....	Rochester, N. Y.

FOR THREE YEARS.

Harry C. Solomon, M. D.....	Boston, Mass.
George H. Kirby, M. D.....	New York, N. Y.

FOR TWO YEARS.

Samuel W. Hamilton, M. D.....	White Plains, N. Y.
Douglas A. Thom, M. D.....	Boston, Mass.

FOR ONE YEAR.

Clarence B. Farrar, M. D.....	Toronto, Canada.
Henry A. Bunker, M. D.....	New York, N. Y.

FOR FIVE YEARS.

Franklin G. Ebaugh, M. D.....	Denver, Colo.
Theophile Raphael, M. D.....	Detroit, Mich.

COMMITTEE ON ARRANGEMENTS.

Herbert C. Woolley, M. D., Chairman.....	Washington, D. C.
Walter L. Treadway, M. D., Vice Chairman.....	Washington, D. C.
Roscoe W. Hall, M. D.....	Washington, D. C.
D. Percy Hickling, M. D.....	Washington, D. C.
Ernest E. Hadley, M. D.....	Washington, D. C.
Lucile Dooley, M. D.....	Washington, D. C.
M. W. Ireland, M. D.....	Washington, D. C.
O. C. Willhite, M. D.....	Washington, D. C.
Ernest L. Bullard, M. D.....	Rockville, Md.
Nell W. Bartram, M. D.....	Washington, D. C.
Ross McC. Chapman, M. D.....	Towson, Md.
Loren B. T. Johnson, M. D.....	Washington, D. C.
Thomas V. Moore, M. D.....	Washington, D. C.

Edward Hiram Reede, M. D.....	Washington, D. C.
Robert R. Dieterle, M. D.....	Washington, D. C.
Lois Dean Hubbard, M. D.....	Washington, D. C.
Amy N. Stannard, M. D.....	Washington, D. C.
Mary O'Malley, M. D.....	Washington, D. C.
Nolan D. C. Lewis, M. D.....	Washington, D. C.
Harriet E. Twombly, M. D.....	Washington, D. C.
John P. H. Murphy, M. D.....	Washington, D. C.

COMMITTEE ON PUBLICITY.

FOR TWO YEARS.

George K. Pratt, M. D., Chairman, 1929-30.....	New York, N. Y.
--	-----------------

FOR FOUR YEARS.

Roger C. Swint, M. D.....	Milledgeville, Ga.
---------------------------	--------------------

FOR THREE YEARS.

A. S. Hamilton, M. D.....	Minneapolis, Minn.
---------------------------	--------------------

FOR ONE YEAR.

Henry B. Elkind, M. D.....	Boston, Mass.
----------------------------	---------------

FOR FIVE YEARS.

Clarence A. Bonner, M. D.....	Boston, Mass.
-------------------------------	---------------

COMMITTEE ON RESEARCH.

FOR FOUR YEARS.

Hugo Mella, M. D., Chairman, 1929-30.....	Northport, N. Y.
---	------------------

FOR THREE YEARS.

Albert M. Barrett, M. D.....	Ann Arbor, Mich.
------------------------------	------------------

FOR TWO YEARS.

Henry A. Bunker, Jr., M. D.....	New York, N. Y.
---------------------------------	-----------------

FOR ONE YEAR.

Arthur S. Hamilton, M. D.....	Minneapolis, Minn.
-------------------------------	--------------------

FOR FIVE YEARS.

John C. Whitehorn, M. D.....	Waverly, Mass.
------------------------------	----------------

COMMITTEE ON STATISTICS.

FOR TWO YEARS.

James V. May, M. D., Chairman 1929-30.....Boston, Mass.
 Albert M. Barrett, Vice-Chairman 1929-30.....Ann Arbor, Mich.

FOR FOUR YEARS.

E. Stanley Abbot, M. D.....Boston, Mass.
 Sanger Brown, II, M. D.....Albany, N. Y.

FOR THREE YEARS.

Phyllis Greenacre, M. D.....White Plains, N. Y.
 William T. Shanahan, M. D.....Sonyea, N. Y.

FOR ONE YEAR.

George H. Kirby, M. D.....New York, N. Y.
 Frankwood E. Williams, M. D.....New York, N. Y.

FOR FIVE YEARS.

C. Macfie Campbell, M. D.....Boston, Mass.
 Walter L. Treadway, M. D.....Washington, D. C.

COMMITTEE ON NURSING.

FOR FIVE YEARS.

Daniel H. Fuller, M. D., Chairman 1929-30.....Philadelphia, Pa.
 Ross McC. Chapman, M. D.....Towson, Md.

FOR FOUR YEARS.

Mortimer W. Raynor, M. D.....White Plains, N. Y.
 Morgan B. Hodskins, M. D.....Palmer, Mass.

FOR THREE YEARS.

Albert Anderson, M. D.....Raleigh, N. C.
 E. H. Cohoon, M. D.....Harding, Mass.

FOR TWO YEARS.

Roger C. Swint, M. D.....Milledgeville, Ga.
 Ralph T. Hinton, M. D.....Elgin, Ind.

FOR ONE YEAR.

Henry I. Klopp, M. D.....Allentown, Pa.
 Earl H. Campbell, M. D.....Newberry, Mich.

COMMITTEE ON STANDARDS AND POLICIES.

FOR TWO YEARS.

George H. Kirby, M. D., Chairman 1929-30.....New York, N. Y.

FOR ONE YEAR.

William M. English, M. D.....Brockville, Ont.

FOR THREE YEARS.

William A. Bryan, M. D.....Worcester, Mass.

FOR FOUR YEARS.

William L. Russell, M. D.....White Plains, N. Y.

FOR FIVE YEARS.

Albert C. Buckley, M. D.....Philadelphia, Pa.

COMMITTEE ON ETHICS.

FOR FOUR YEARS.

George H. Kirby, M. D., Chairman 1929-30.....New York, N. Y.

FOR THREE YEARS.

Albert M. Barrett, M. D.....Ann Arbor, Mich.

FOR TWO YEARS.

George A. Johns, M. D.....Jefferson City, Mo.

FOR ONE YEAR.

Edward N. Brush, M. D.....Baltimore, Md.

FOR FIVE YEARS.

George S. Adams, M. D.....Yankton, S. D.

COMMITTEE ON LEGAL ASPECTS OF PSYCHIATRY.

FOR FOUR YEARS.

George M. Kline, M. D., Chairman 1929-30.....Boston, Mass.

L. Vernon Briggs, M. D.....Boston, Mass.

FOR THREE YEARS.

Karl A. Menniger, M. D.....Topeka, Kans.

William A. White, M. D., Vice-Chairman 1929-30.....Washington, D. C.

FOR TWO YEARS.

Frankwood E. Williams, M. D. New York, N. Y.
 Herman M. Adler, M. D. Chicago, Ill.

FOR ONE YEAR.

Winfred Oversholser, M. D. Boston, Mass.
 Raymond F. C. Kieb, M. D. Albany, N. Y.

FOR FIVE YEARS.

Bernard Glueck, M. D. Ossining, N. Y.
 William Healy, M. D. Boston, Mass.

COMMITTEE ON MEDICAL SERVICES.

FOR THREE YEARS.

William C. Sandy, M. D., Chairman 1929-30. Harrisburg, Pa.
 Mortimer W. Raynor, M. D., Vice-Chairman 1929-30. White Plains, N. Y.

FOR FOUR YEARS.

Edward A. Strecker, M. D. Philadelphia, Pa.
 Lloyd J. Thompson, M. D. New Haven, Conn.

FOR TWO YEARS.

William Rush Dunton, M. D. Catonsville, Md.
 G. Kirby Collier, M. D. Rochester, N. Y.

FOR ONE YEAR.

Glenn E. Myers, M. D. Los Angeles, Cal.
 Ransom A. Greene, M. D. Waverly, Mass.

FOR FIVE YEARS.

Frederick W. Parsons, M. D. Albany, N. Y.
 Karl M. Bowman, M. D. Boston, Mass.

COMMITTEE ON RELATIONS WITH SOCIAL SCIENCES.

FOR FIVE YEARS.

William A. White, M. D., Chairman 1929-30. Washington, D. C.

FOR FOUR YEARS.

Arthur H. Ruggles, M. D. Providence, R. I.

FOR THREE YEARS.

George M. Kline, M. D. Boston, Mass.

FOR TWO YEARS.

Harry Stack Sullivan, M. D., Secretary.....Towson, Md.

FOR ONE YEAR.

C. Floyd Haviland, M. D.*.....New York, N. Y.

COMMITTEE ON PSYCHIATRIC SOCIAL SERVICE.

FOR FIVE YEARS.

Karl M. Bowman, M. D., Chairman 1929-30.....Boston, Mass.

FOR FOUR YEARS.

George H. Stevenson, M. D.....New York, N. Y.

FOR THREE YEARS.

J. Allen Jackson, M. D.....Danville, Pa.

FOR TWO YEARS.

Guy Payne, M. D.....Cedar Grove, N. J.

FOR ONE YEAR.

George M. Kline, M. D.....Boston, Mass.

COMMITTEE ON ACTIVITIES OF THE NEUROPSYCHIATRIC
DIVISION OF THE VETERANS' BUREAU.

Glenn E. Myers, M. D., Chairman 1929-30.....Los Angeles, Cal.

George M. Kline, M. D.....Boston, Mass.

Albert M. Barrett, M. D.....Ann Arbor, Mich.

COMMITTEE ON GRADUATE EDUCATION IN PSYCHIATRY.

Dr. Adolf Meyer, Chairman.....Baltimore, Md.

Dr. George H. Kirby.....New York, N. Y.

Dr. Edward A. Strecker.....Philadelphia, Pa.

THE EIGHTY-SIXTH ANNUAL MEETING OF THE AMERICAN PSYCHIATRIC ASSOCIATION.—The next annual meeting of the Association, the eighty-sixth, will be held in Washington, May 5-9, 1930. The headquarters of the Association, as well as the place of meeting, will be at the Willard Hotel.

As is generally known The First International Congress on Mutual Hygiene will be held in Washington, May 5-10, 1930. The

* Deceased.

American Association for the Study of the Feeble-Minded will hold its annual meeting in Washington during the same week. The bureau and headquarters of the International Congress will be at the Willard Hotel.

In view of the foregoing facts we deem it our duty to call the attention of members of our Association intending to attend the meeting in May to the necessity of making early reservations for rooms.

There are other hotels near the Willard and the Chairman of the Committee of Arrangements, Dr. Herbert C. Woolley, St. Elizabeths Hospital, Washington, D. C., will advise any member, unable to obtain reservations at the Willard, as to the other available accommodation. We advise prompt action.

SECOND INTERNATIONAL CONGRESS FOR SEX RESEARCH.—The Second International Congress for Sex Research will be held in the House of the British Medical Association, Tavistock Square, London, August 3 to 9, 1930, under the presidency of Professor F. A. E. Crew, of Edinburgh. Both in membership and in importance it is expected that this meeting will notably excel the First International Congress, held in Berlin in October, 1926. Those who are interested are requested to write Professor Crew, The University, West Mains Road, Edinburgh.

The organization and purposes of the congress can be fully served only by having all of the many aspects of sex research properly represented among the papers read at the London meeting. To American workers it is highly desirable that American investigations and investigators should be adequately represented at London, particularly since few persons from this country attended the first congress.

An American committee has been formed for the purpose of securing a maximum participation in the congress by workers in this country. The members of this committee, representing biology, medicine, psychology, sociology and anthropology, are: Dr. Oscar Riddle, chairman, Dr. A. A. Brill, Dr. Calvin P. Stone, Dr. William F. Ogburn and Dr. Clark Wissler.

NOTICE.—The Committee on the Family of the Social Science Research Council is endeavoring to collect copies of questionnaires, schedules and other blanks used by investigators studying

the family. It is proposed to establish this material as a loan collection in the office of the Social Science Research Council in New York City, so that responsible institutions and investigators may borrow the collection for a limited period when organizing research plans.

The cooperation of all investigators in the field of the family is earnestly requested, since a fairly complete collection is desired, and you are asked to send immediately, *three complete sets* of all such forms appropriately marked to show the institution and the name of the investigator to the Social Science Research Council, 230 Park Avenue, New York City.

AMERICAN ORTHOPSYCHIATRIC ASSOCIATION.—The Seventh Annual Meeting of the American Orthopsychiatric Association will be held at the Hotel Pennsylvania, New York, on February 21 and 22, 1930. The president is Dr. Lawson G. Lowrey, and the Secretary, Dr. George S. Stevenson, 370 Seventh Ave., New York.

Book Reviews.

Bodily Changes in Pain, Hunger, Fear and Rage. By WALTER B. CANNON.
404 pages. (New York: D. Appleton & Co., 1929.)

This second edition of the author's well-known volume brings it up to date and enormously increases its value. There has been a great deal of new material added, and some of this is of particular interest to the psychiatrist.

Chapter I is entitled "The Effect of the Emotions on Digestion." This forms an excellent discussion of the emotions which are favorable and unfavorable to the digestive process, with illustrations from animal experimentation.

Chapter II is entitled "The General Organization of the Visceral Nerves Concerned in Emotions." This particularly discusses the antagonistic action of the sympathetic division to both the cranial and sacral divisions, and points out that stimulation of the sympathetic division has many of the same effects as stimulating the adrenal glands.

The discussion of the secretion of adrenalin then follows.

After this is a discussion of "The Increase of Blood Sugar in Pain and Great Emotion." The relationship of fatigue and adrenin, and activity of the adrenal gland with results and effects of blood pressure, and the coagulation time of the blood is gone over in detail.

One chapter is devoted to "Emotional Increase of Red Blood Corpuscles." Cannon finds that emotional excitement produces an increase averaging 27 per cent in the erythrocyte count. He believes this is dependent on the nervous control of the spleen. He also adds that a relative mononucleosis of the white blood corpuscles is produced by excitement.

The last half of the book is devoted largely to the interpretation of this material.

It seems unnecessary to repeat the fundamental points concerning the protective nature of these bodily changes in emotional states which appeared in the first edition.

However, it does seem worthwhile to point out the further developments which Cannon has made. Cannon emphasizes the localization of a center in the optic thalamus which is of great importance in the emotional life of the individual. He believes that some of the skeletal muscles have both a cortical and thalamic control, so that laughter, for example, may be thalamic—as when we laugh at a ludicrous situation, or it may be cortical—as when we laugh voluntarily. He points out that the viscera are under purely thalamic control, and that no cortical or voluntary act can influence their behavior except in some indirect manner.

The three chapters entitled "The Inter-Relations of Emotions," "Critical Examination of the James-Lange Theory of Emotions," and "Emotion as a

Function of the Optic Thalamus" should be carefully studied by every psychiatrist. Cannon rejects the James-Lange theory. Briefly stated, he thinks of the optic thalamus as the emotional center from which impulses pass to the cortex of the brain, giving rise to the conscious feelings of emotion, and from which other impulses pass to the viscera, giving rise to the physiological expression of emotion. In rejecting the James-Lange theory he points out certain defects and contradictions in it.

In the reviewer's opinion, while Cannon's arguments appear sound, there is no point made of the possibility that impulses from the viscera to the optic thalamus may produce emotion on the basis of the well-known conditioned reflex. If Cannon's theory is true, it would seem obvious that a conditioned reflex would be quickly established so that whenever the physiological accompaniments of emotion are produced the emotion will tend to recur for this reason. This may be the essential truth of the James-Lange theory, and the reason why, as James puts it, that if we grit our teeth and frown we tend to become angry, an observation which is familiar to almost every one, and which can be easily verified.

The book closes with a chapter entitled "Alternative Satisfaction for the Fighting Emotions," in which he raises the question as to whether the fighting emotions may not be sublimated in the form of athletic rivalries and thus aid in doing away with war.

In closing the reviewer cannot too strongly emphasize his feeling that every psychiatrist should read this book and be familiar with its contents. It contains a great deal of fundamental work which should be known to every person dealing with emotional problems.

KARL M. BOWMAN,
Boston Psychopathic Hospital.

Epilepsy. By L. J. J. MUSKENS, M. D. Foreword by SIR CHARLES S. SHERINGTON, M. D. (New York: William Wood and Company: 1928; 435 pp.)

This English edition of the author's work on epilepsy is a volume of some 430 pages. It is divided into three parts; the first considers experimental research on the myoclonic reflex and myoclonic epileptic seizures. This is the particular field in which the author's views find their expression. "There is a group of reflexes which has been little studied and yet is common among undomesticated warm-blooded animals. . . . They are elicited by some unexpected touch or unexpected noise. . . . It is interesting to find that a normal epileptic reaction . . . corresponds with the phenomenon here described as a myoclonic reflex. . . . It seems difficult therefore to explain a myoclonic epileptic fit except as a method of discharging an organism from the influence of a toxic substance. . . . Physiology teaches little about the structures in the central nervous system which we have as centers for these reinforced myoclonic reflex convulsions. . . . Whether the phenomena concomitant to an epileptic fit . . . are of significance in discharging the central nervous system, I am not prepared to say. . . . These

physiological observations give the impression that in incipient myoclonic epilepsy we deal with a physiologic condition which leads to a pathologic one. . . . Viewed from this standpoint the epileptic phenomena come into line with other reflex phenomena with a physiologic basis, such as inflammation, pyrexia, cold shivering, or rigors. These latter reflexes are fundamentally protective, although ultimately they emerge into well-recognized dangerous pathological conditions. I submit that an epileptic fit is essentially a protective measure on the part of the central nervous system. The epileptic fit . . . is from the first a potentially pathological condition of great danger to the organism."

Part II includes seven chapters dealing with "the influence of lesions in the central nervous system on the myoclonic reflexes and myoclonic epileptic fits." "The object of the second part of this book has been to establish as accurately as possible the significance of these fits in the physiological economy and to ascertain how far localization of these phenomena in the central nervous system is possible."

Part III deals in fifteen chapters with "epileptic disorders observed in man, and their treatment." A classification is offered, "based on the etiology and symptoms of the disorder"; this includes (a) myoclonic epileptic fits elicited by well-known toxins, (b) epileptic fits caused by certain toxins produced endogenously, (c) cases of epilepsy due to some local affection of the brain, not to trauma, (d) traumatic epilepsy. The author gives also a brief classification based on etiology and another based on symptoms. The latter is (1) parial or Jacksonian epilepsy, (2) primary grand mal, (3) primary petit mal, and (4) primary myoclonic epilepsy. His fourth classification is based on the period of life at which the disorder appears. Chapter headings in Part III are: Classification of Epilepsy; Myoclonic Epilepsy; A More Detailed Description of Regional and Myoclonic Shocks in Man; Common or Genuine Epilepsy with Myoclonic Epilepsy as the Starting-Point: Origin and Course; Tonic Spasms and Convulsions in Childhood and Their Significance for the Development of Epilepsy; Tonic Spasms, Etc.; The Influence of Menstruation and Pregnancy on the Occurrence of Epilepsy; The Further Description of the Symptoms of Epilepsy and Their Significance; The Relative Frequency and Nosological Value of the Symptoms; The Statistics of Epilepsy; Differential Diagnosis of Epilepsy; Fainting Fits and Psychisms; Treatment; Pathological Anatomy; Traumatic Epilepsy and Surgical Treatment; A System for the Prevention of Epilepsy, and for the Care of Epileptics. The discussion is rather well illustrated with excerpts from clinical records; there are an abundance of tabulations, well printed photomicrographs, and a good author and subject index.

HARRY STACK SULLIVAN,

Sheppard and Enoch Pratt Hospital.

In Memoriam.

DR. C. FLOYD HAVILAND.

Friends and associates of Dr. C. Floyd Haviland, superintendent of the Manhattan State Hospital, Ward's Island, New York City, and member of the Executive Committee of the National Committee for Mental Hygiene, have been shocked to learn of his death in Cairo, Egypt, on the morning of January 1. Dr. and Mrs. Haviland left New York on December 3 for a Mediterranean cruise and they spent Christmas Day in Jerusalem. From reports obtained up to the present writing, it appears that Dr. Haviland developed influenza pneumonia shortly after arriving in Cairo. He was under the care of several American physicians in the Anglo-American Hospital, where he died. It is expected that funeral services will be held at Ward's Island upon the return of Mrs. Haviland and the remains to this country; the date is as yet undetermined.

Dr. C. Floyd Haviland was born on August 15, 1875, in Spencer-town, N. Y., the son of Dr. Norman H. Haviland, and the late Mrs. Henrietta Newman Haviland. The family later moved to Fulton, N. Y., where Dr. Haviland attended the public schools, being graduated from the high school in 1893. He received his medical education at the Syracuse Medical School, obtaining his degree in 1896.

In 1897 Dr. Haviland joined the staff of the Manhattan State Hospital and passed through the grades of medical interne, junior physician, and second assistant physician, serving under Dr. William Mabon. In 1910 he was promoted to first assistant physician at the Kings Park State Hospital on Long Island, where he remained until 1915, when he became superintendent of the Connecticut State Hospital at Middletown, Conn.

While at King's Park, in 1914, Dr. Haviland, at the request of the National Committee for Mental Hygiene, made a comprehensive survey of the care of the insane in Pennsylvania. From 1916 to 1921 he was chairman of the Executive Committee of the Connecti-

cut Society for Mental Hygiene, and in 1921 he was president of the Connecticut Conference on Social Work.

On December 19, 1921, Dr. Haviland became medical member and chairman of the New York State Hospital Commission, in which position he continued until he resigned on July 1, 1926, to become superintendent of the Manhattan State Hospital. At the end of Dr. Haviland's service as head of the State Hospital Commission it was remarked that his period of service had covered an epoch in the development of the state system in the care and treatment of the mentally diseased, and in no equal period of time since the passage of the State Care Act in 1890 had so many progressive measures been taken by the Commission. In his position as chairman and chief executive of the Commission Dr. Haviland had had two purposes continually in mind: First, the improvement of the state hospitals, so that more patients might recover or improve; second, the prevention of mental diseases, so that fewer patients would need state hospital treatment. For the accomplishment of the first of these purposes action was taken during his service as a commissioner in organizing and developing occupational therapy in all of the state hospitals; a medical survey of the state hospitals was made and the medical services placed on more uniform and efficient basis, and diagnostic clinics in several of the state hospitals were organized; a survey of nursing and the schools of nursing in the state hospitals was made and steps made to strengthen and improve the service and to provide better courses of instruction for both nurses and attendants; as a result of the approval of the bond issue for fifty million dollars for state institutions a comprehensive building program to remove overcrowding and to provide better facilities for patients was undertaken; two new state hospitals were established, a veterans memorial hospital was built, and extensive development was carried on in the institutions previously begun at Marcy and Creedmore; for the protection from fire in the state hospitals extensive repairs were made and sprinkler systems and other protective devices were installed in the state hospitals. To accomplish the second of the purposes above mentioned Dr. Haviland was active in increasing the mental clinics conducted by the state hospitals, the number of social workers was increased and efforts were made to extend the activities of the hospital clinics to problem children. Dr. Haviland was intensively

interested in bringing about the provision for the erection of the new State Psychiatric Institute and Hospital, in connection with the Medical Center, New York City, and also in the formulation of plans for the erection of a psychiatric hospital to form a part of the medical center of Syracuse University. Dr. Haviland believed that these two institutions represented the crowning achievement of his work on the Commission.

In the last few years while superintendent of the Manhattan State Hospital, Dr. Haviland had continued his energetic activities in, and enthusiastic support of, matters relating to the better care of mental patients, and to mental hygiene problems in the community. He was active not only in the National Committee for Mental Hygiene, but also was a member of the Mental Hygiene Committee of the State Charities Aid Association, and of the New York City Mental Hygiene Committee. A Member and Fellow of the American Psychiatric Association since 1899, Dr. Haviland served as Secretary-Treasurer of that Association from 1921 to 1924, was Vice-President in 1925 and was President in 1926. The interests of that Association were always close to his heart, and his activity in it did not cease with his retirement from the presidency, but continued to show itself in the subsequent work on the Executive Committee and on the Committee on the Relations with Social Sciences, of which he was a member at the time of his death. Likewise at the time of his death he was President of the New York Society for Clinical Psychiatry. He was Associate Editor of "The Modern Hospital."

Dr. Haviland's wide and varied interests were further shown in his membership and active participation in the following organizations: American Association for the Advancement of Science, American Genetic Association, Association for Research in Mental and Nervous Diseases, Eugenics Research Association, American Psychopathological Association, American Social Hygiene Association, American Neurological Association, American Eugenics Association, New York State Occupational Therapy Association, New York Neurological Society, New York Psychiatric Society, New York Society of Medical Jurisprudence, Phi Kappa Psi, Nu Sigma Nu, and Phi Kappa Phi fraternities and the Lotos, International, and Medical clubs.

Dr. Haviland was formerly clinical assistant in the departments of neurology and psychiatry in the College of Physicians and Surgeons of Columbia University; since 1927 he had been clinical professor of psychiatry in that school.

Dr. Haviland is survived by his widow, his father, and by a brother Dr. F. Ross Haviland, first assistant physician at the Brooklyn State Hospital.

To know Floyd Haviland was a delight and a pleasure; to be associated with him in his professional work was a privilege. Always enthusiastic and receptive toward progressive ideas looking toward the welfare not only of the mental patients, but of mankind in general, his enthusiasm was a stimulus to those with whom he worked. Genial and kindly in disposition he imbued his friends and associates and patients with an optimistic spirit; difficulties did not terrify him, but rather stimulated him to greater and usually successful feats of accomplishments. Indefatigable in work, no task or new activity was too great for him to attempt and increasing demands upon his time and effort were never met by refusals. He gave his life to his work; possibly his life would have been longer if he had considered himself more, but those who knew him well realize that to him a longer life with inactivity would have held none of the satisfaction that his all too short active life gave him.

To the members of his family privileged to have him as a son, husband and brother, the heartfelt sympathy of Floyd Haviland's friends is extended. "That last day does not bring extinction to us, but change of place."

CLARENCE O. CHENEY.

DR. EDGAR O. CROSSMAN.

Dr. Edgar O. Crossman, a member of the Massachusetts Psychiatric Society, died at his home in Bedford, New Hampshire, on June 21, 1929, after a brief illness.

Dr. Crossman was born in Vermont on December 15, 1864, and graduated from the Medical School of the University of Vermont in 1887. He practised both in Vermont and New Hampshire, and served as a major in the Medical Corps of the Army during the World War. Upon his discharge from the Army, he became connected with the United States Veterans Bureau, which he served

with distinction until the time of his death. He was for a time district manager of the New England District, and became medical director of the bureau in April, 1924. In July, 1926, at his request, he was transferred from Washington to Boston to become medical officer in charge of the U. S. Veterans Hospital at West Roxbury. While here he also supervised the construction of the Veterans Hospitals at Northport, L. I., and Bedford, Mass. In 1928, upon the urgent solicitation of his chief, General Hines, and at a considerable personal sacrifice, he returned to Washington as medical director. He was actively engaged in his official duties when stricken with his fatal illness. He was buried with full military honors in Arlington Cemetery.

Dr. Crossman will be long remembered by those who had the privilege of knowing him as a loyal, genial friend. To his patients, his memory remains as that of a devoted, faithful physician. To the country at large, he stands as one who did much to place the medical work of the Veterans Bureau upon a high plane, and who labored unremittingly to secure to the disabled veterans of the World War hospital care of the highest quality.

The Massachusetts Psychiatric Society desires to place permanently on record its sense of loss in the passing of a distinguished member and to express to Dr. Crossman's widow and son its deepest sympathy.